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Oral Surgery and Radiography

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the Sciences of Orthodontia, Oral Surgery
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VOL. XI

ST. LOUIS, FEBRUARY, 1925

No. 2

ORIGINAL ARTICLES

METHOD OF PRESENTING CORRECT DIET TO ORTHODONTIA PATIENTS*

BY HENRY F. HOFFMAN, D.D.S., DENVER, COLORADO

THE charts shown here are designed to assist our patients in the selection of a normal diet conforming to the best scientific information available at this time. They are arranged to cover the subject in a condensed and logical manner, the object being to convey the necessary information plainly and with the fewest words possible. We explain that we do not prescribe diets for diseased conditions.

We go over these charts with our older patients and with the parents of our younger patients showing only one at a time, generally using a half-hour to an hour explaining and answering questions.

I no longer start orthodontic treatment for patients presenting marked evidence of malnutrition until I have the definite cooperation of the parents in the matter of diet, and then in the worst cases, I delay treatment until there is some marked improvement in the child. Such a policy insures continued cooperation and can reasonably be expected to shorten the time of treatment and retention, and works for a better final result.

The time consumed in this work is more than compensated for by the saving in time and energy resulting from the cooperation of parents and patients, and the improved physical condition of the patients. Malnourished children are usually finicky, irritable and generally hard to work for and are subject to more than the ordinary amount of sickness, all of which is largely corrected or completely eradicated when normal nutrition is secured.

The first chart (Fig. 1), gives the five classes of foods necessary to ideal nutrition with their functions and ideal sources.

*Given before the American Society of Orthodontists, Kansas City, Mo., March 18-21, 1924.

TO HAVE HEALTH

Perfect skeletal structure--Perfect muscle development-- Normal energy
Resistance to disease-- Long life

The dietary **MUST** contain the required amounts of the following five classes of foods:

	<u>Functions</u>	<u>Ideal Sources</u>
PROTEINS	{ Build Repair	{ Meat, Fish, Legumes, Cottage cheese, and MILK
CARBOHYDRATES	{ Furnish heat and energy	{ Cereal grains, Potatoes and MILK
FATS	{ Furnish heat and energy	{ Vegetable oils, Butter fat, and MILK
MINERAL SALTS	{ Build bone and teeth	{ Non-starchy vegetables, Glandular meat, and MILK
VITAMINS	{ Protect from disease Necessary to growth	{ Fruit, Raw vegetables Glandular meat, and MILK

Fig. 1.

TYPES of MEALS and FOOD COMBINATIONS

Have one **PROTEIN** meal a day. Have one **CARBOHYDRATE** meal a day.

This will avoid overeating. Your energy will be used for growth, not digestion.

AVOID { Two proteins at same meal.
Two carbohydrates at same meal.
Milk with meat.

HAVE { One cooked non-starchy vegetable with every protein and every carbohydrate meal.
Fruits and raw vegetable salads with protein meals.
Sweet fruits and raw vegetable salads with carbohydrate meals.

The **KEYNOTE** of an **EXCELLENT MEAL** is
SIMPLICITY

Fig. 2.

FOOD and the TEETH

Preventive Dentistry should begin with scientific nutrition in prenatal life and infancy.

The principal components of the teeth are calcium and phosphorus.

Diet of the expectant mother must contain a generous quantity of calcium and phosphorus foods.

Diet requirements, for bone and tooth building, are the same after birth.

Formation of deciduous teeth begins at the 17th week of embryonal life.

Formation of permanent teeth begins at the 25th week of embryonal life.

At birth (40th week) crowns of all deciduous teeth and "six-year molars" are largely formed.

Fig. 3.

MILK

Milk is the most satisfactory single article of food suitable for consumption by man.



It is not ideal taken over a long period of time as the sole source of nutriment.

Milk supplements and corrects all the deficiencies of other foods.

In pasteurization, one Vitamin is destroyed. This may be supplied by eating oranges, tomatoes, lemons or grapefruit.

Milk is deficient in iron. This may be supplied by eating spinach or raisins.

Milk should not be combined with meat.

Every child should have a quart of milk every day.

Fig. 4.

THE PROTEINS

Lean Meat Fish Fowl Game Nuts Legumes

The **P**roteins build and repair body tissues and cells.

An excess of protein is eliminated at the expense of the kidneys.

Meat is a stimulant. In excess it is like a drug.

Pork is difficult to digest.

Aside from breast of chicken and game, lamb is the easiest to digest.

Glandular meats, sweetbreads, kidney, liver, brains, are the best quality of protein.

LEGUMES Dried beans Peas Peanuts.

These are a poor quality of protein.

Equivalent of concentrated meat and bread (protein and carbohydrate)

Usually eaten in quantities beyond bodily requirements.

Do not eat with meat, nuts or cheese.

Fig. 5.

COOKING MEAT

Pan broiling. Have the pan hot enough to turn meat white when put into pan. Instantly turn meat over and repeat four times. Then cook slower until done.

Meat for broiling should be at least one inch thick.

Round steak. **S**ear in very hot pan. Then add a small amount of water and simmer until tender.

Large roasts are very excellent.

Stewing meat. **N**ever boil violently, simmer throughout process.

Fig. 6.

MEAT SUBSTITUTES

EGGS

Should never be eaten when meat is eaten.

The same combinations with meat are correct combinations with eggs.

NUTS

Should be substituted for meat at meat meal and never in excessive quantities.

Most nuts should be blanched. The skin is irritating to the lining of the stomach.

COTTAGE CHEESE

Cottage cheese is the best cheese known.

An ideal substitute for meat.

One of the best building, or protein foods.

Fig. 7.

CARBOHYDRATES

Foods which produce heat and energy

Bread - Cereal - Potatoes



BREAD

Should always be of whole wheat or whole grain flour.

Should be thoroughly baked.

Do not eat with meat meal. It makes an excess of concentrated food.

Do not eat with acid fruits. The starch and acid set up a fermentation.

Bread toasted is the most wholesome way. It is harder to chew making insalivation unavoidable.

Fig. 8.

CEREALS

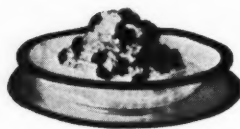
WHOLE WHEAT

Cook in double boiler two hours, or more.

It should be a stiff consistency, not a porridge.

If sweetening is necessary use raisins or figs, never white sugar.

A very small quantity of dark brown sugar may be used occasionally.



OLD FASHIONED OATMEAL

Cook to thick consistency. A mushy porridge will not be insalivated.

Bread should not be eaten with cereal.

Acid fruit should not be eaten with cereal.

Dairy products, butter, cream, milk, combine well with cereals.

"Shredded Wheat" and "Triscuit" contain all of the wheat and are excellent.

Fig. 9.

POTATO

White Potato --- Yam --- Sweet Potato

These are the most important carbohydrate or starch foods.

They should be baked, steamed or boiled, never fried.

These are easily digested, very nutritious and not acid forming.

Always serve non-starchy vegetables when potatoes are used.

SUGAR

Produces heat and energy (same as the starches).

Natural sugars, found in fruit and root vegetables, are indispensable.

Refined white sugar should never be used. It satisfies the appetite without giving nourishment to the body.

It leaves a substance in the mouth which produces decay of the teeth.

Interferes with digestion of other carbohydrates permitting fermentation.

Eat raisins, figs, and dates instead of candy.

Fig. 10.

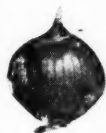
FATS**Butter****Fat meat****Olive oil****Cream****Fat fish****Peanuts****Cheese****Nuts****Cocoanuts**

Fats produce heat and energy

Butter and cream are the best fat foods.

Fat, being a heat producing food should be eaten sparingly during summer months.

Fig. 11.

**MINERAL SALTS and VITAMINS
NON-STARCHY VEGETABLES**

Furnish food salts necessary to health and growth.

Should give much of the bulk to the meal.

May be combined with any other foods.

**Artichoke (French)****Egg plant****Chives****Brussel Sprouts****Sweet peppers****Parsnips****Pumpkins****Beet tops****Fresh green peas****Asparagus****Onions****Rutabagas****Fresh green beans****Beets****Squash****Turnip tops****Cauliflower****Carrots****Turnips****Swiss Chard****Green corn****Celery****Garlic****Spinach****Raw Vegetables To Be Used For Salads**

Raw fruit or raw vegetables **MUST** be included in every MEAT meal.

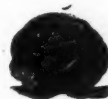
Carrots**Sweet peppers****Radishes****Onions****Water Cress****Cucumbers****Cabbage****Garlic****Lettuce****Parsley****Celery****Chives**

Fig. 12.

COOKING VEGETABLES

Do not cook with so much water that it must be drained before serving.

Any surplus water should be served in soup or with the vegetable. It contains important mineral salts.

Add no salt or seasoning while cooking.

Have tight fitting lid, and heat for simmering, not violent boiling.

Steam cooking is the best way to cook vegetables. All the salts are retained.

A double boiler for cooking vegetables is excellent. It takes less water, and reduces the danger of burning.

Fig. 13.

FRESH FRUIT

Fruit is not a luxury, but a vital necessity in the diet.



Fruit contains an abundance of food salts, sugar, and vitamins.

Raw fruit is the greatest enemy to the disease known as Scurvy.

Cooking throws the mineral salts out of harmony and destroys Vitamin C.

Acid fruits combine with anything but starches.

Lemons	Apricots	Mulberries	Tomatoes
Limes	Apples	Huckleberries	Blackberries
Oranges	Pears	Elderberries	Cranberries
Grapefruit	Peaches	Black Currants	Gooseberries
Pineapple	Grapes	Plums	Loganberries
Currants	Cherries	Prunes	Strawberries
			Raspberries

Fig. 14.

DRIED ACID FRUITS

DRIED SWEET FRUITS

If dried fruit is used know that it is dried without chemicals.

Apricots

Raspberries

Dates

Raisins

Peaches

Loganberries

Figs

Sweet Prunes

Plums

Blackberries

Prunes

Currants

Figs, dates, and raisins are the best of the sweet dried fruits.

Apples

Pears

MELONS

Muskmelon

Cantaloupe

Casaba

Watermelon

Should never be eaten unless digestion is sound.

Ferment quickly when eaten with other food.

Combine with nothing but fresh fruit.

Fig. 15.

VITAMINS

Vitamins are chemical compounds which are present in natural foods grown in the sunlight.

They are absolutely necessary for proper growth, maintenance, and protection from disease.

They are destroyed by cooking at high temperatures.

A well balanced diet should contain a sufficient number of all vitamins.

A restricted or poorly selected diet may be deficient in one or all of the vitamins.

Fig. 16.

The second chart (Fig. 2), gives some very simple rules covering types of meals and food combinations. The entire subject of proper combinations of food is covered in this chart under the two specified headings "Avoid" and "Have," with three rules under each heading.

At least one meal a day should contain protein as the leading ingredient and one meal should contain considerable carbohydrate but the proper combinations should be used with the protein and the carbohydrate meals. Some combinations are undesirable as "two proteins at the same meal" because they are not all digested. The body can accommodate and digest only a limited amount of protein.

Teaching Children To Eat Wholesome Foods

Begin from babyhood to cultivate taste for important foods.

Parents must eat cheerfully all foods that the child should have.

Never talk of food dislikes; children will imitate those dislikes.

Serve only palatable, fresh and well cooked foods.

Give a new food as one that the child is now big enough to have.

Keep diet simple; few sweets; few foods of too distinctive flavor.

Use authority if necessary. **B**ut, development of real liking by other six methods is far better.

A notional appetite can be most easily corrected in a nutrition class.

Fig. 17.

Two carbohydrates at the same meal should be avoided because it gives too much concentrated food to the exclusion of the foods containing roughage, mineral salts, and vitamins. Meat and milk in combination give two proteins. Cooked nonstarchy vegetables should be eaten with every protein and every carbohydrate meal to supply the mineral salts and roughage. Fruits and raw vegetable salads with the protein meals supply vitamins, mineral salts and roughage. Sweet fruits *only*, and raw vegetable salads should be used at the carbohydrate meals. The acid of the acid and subacid fruits sets up a fermentation when combined with carbohydrates.

We explain with great care the chart on "Food and the Teeth" (Fig. 3), to impress the relation of proper food to the formation of good tooth and

bone structure. The necessity for proper quantities of foods containing the "Mineral Salts and Vitamins" (Fig. 12) is explained.

In the chart on "Fresh Fruit" (Fig. 14) great emphasis is put on not

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A Guide for Planning Menus

Arranged by Henry F. Hoffman, D. D. S., and Ethel Covington, D. H.

TO HAVE HEALTH

Perfect skeletal structure—Perfect muscle development—Resistance to disease—
Normal energy—Long life.

The dietary **MUST** contain the required amounts of the following five classes of foods:

FUNCTIONS	IDEAL SOURCES
1. PROTEINS { Build Repair	{ Meat, Fish, Legumes Cottage Cheese, and Milk
2. CARBOHYDRATES .. { Furnish heat and energy	{ Cereal Grains Potatoes and Milk
3. FATS { Furnish heat and energy	{ Vegetable Oils, Butter Fat, and Milk
4. MINERAL SALTS { Build bone and teeth	{ Non-Starchy Vegetables Glandular Meat, and Milk
5. VITAMINS { Protect from disease Necessary to growth..	{ Fruit, Raw Vegetables Glandular Meat, and Milk

TYPES OF MEALS AND FOOD COMBINATIONS

Have one **PROTEIN** meal a day. Have one **CARBOHYDRATE** meal a day.
This will avoid overeating Your energy will be used for **GROWTH**, not digestion.

AVOID

{

Two proteins at same meal.
Two carbohydrates at same meal.
Milk with meat.

HAVE

{

Cooked non-starchy vegetables with every protein and every carbohydrate meal.
Fruits and raw vegetable salads with protein meals.
Sweet fruits and raw vegetable salads with carbohydrate meals.

The **KEYNOTE** of an **EXCELLENT MEAL** is **SIMPLICITY**

(Over)

Fig. 18.—(Obverse side.)

combining acid and subacid fruits with carbohydrates. They may be combined with milk if carbohydrates are not present in any form. This very bad practice of combining acid fruits and carbohydrates is an almost uni-

versal custom. Tomatoes, it should be noted, are classed with the acid fruits.

We call especial attention to the remarks on melons (Fig. 15) which should receive more general recognition. We go into the subject of "Vita-

<p>PROTEINS</p> <p>Lean Meat Fish Fowl Game Nuts Legumes</p> <p>LEGUMES</p> <p>Dried Beans Dried Peas Peanuts</p> <p>MEAT SUBSTITUTES</p> <p>Eggs Nuts Cottage Cheese</p> <hr/> <p>CARBOHYDRATES</p> <p>Bread Cereals Potatoes</p> <hr/> <p>FATS</p> <p>Butter Cream Fat Meat Fat Fish Vegetable Oils</p> <hr/> <p>MINERAL SALTS AND VITAMINS</p> <p>RAW VEGETABLES TO BE USED FOR SALADS</p> <p>Carrots Water-cress Lettuce Sweet Peppers Cucumbers Parsley Radishes Cabbage Celery Onions Garlic Chives</p>	<p>NON-STARCHY VEGETABLES</p> <p>Artichoke (French) Sweet Peppers Fresh Green Peas Fresh Green Beans Cauliflower Green Corn Egg Plant Parsnips Asparagus Beets Carrots Celery Chives Pumpkin Onions Squash Turnips Garlic Brussels Sprouts Beet Tops Rutabagas Swiss Chard Spinach</p> <p>FRESH FRUIT</p> <p>Lemon Lime Orange Grapefruit Pineapple Currants Apricots Apple Pear Peach Grapes Cherries Mulberries Huckleberries Elderberries Plums Prunes Blackberries Cranberries Gooseberries Loganberries Strawberries Raspberries Tomatoes</p> <p>DRIED SWEET FRUITS</p> <p>Dates Figs Raisins</p>
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Fig. 19.—(Reverse side of Fig. 18.)

mins'' (Fig. 16) very briefly, merely explaining that they are absolutely necessary to growth and protection from disease but that in a properly arranged diet all the vitamins are present in ample quantities.

The last chart, "Teaching Children to Eat Wholesome Food" (Fig. 17), we show to parents only. Most food likes or dislikes are purely matters of imitation or habit and parents can only expect to control the appetite habits of their children when they themselves create the proper mental attitude by the right example. We make a great deal of this chart often suggesting to the parents a line of reading on child psychology which when followed up proves of inestimable value to all concerned.

Used together, Charts Nos. 1 and 2 form a guide for meal planning. They are printed on one card of suitable size to hang up in the kitchen (Fig. 18). On the reverse side (Fig. 19) is a printed list of the principal articles of food from which each of the general classes of foods are derived. Since the source of mineral salts and vitamins are chiefly the same, these foods are classed under one heading, "Mineral Salts and Vitamins."

A diet conforming to these few simple rules will promote normal health and development. Anyone should be able to understand them even without a knowledge of chemistry. The classifications of articles of food on the back of the card (Fig. 19) enables the housewife to use beneficially almost everything offered for sale as food, excepting the refined and denatured prepared foods such as refined sugars and syrups, white flour and the other milled cereals which should be permanently eliminated from our diet.

The diet to best promote and maintain normal nutrition is neither monotonous nor restricted, but is almost entirely a matter of proper combinations of food.

To Miss Ethel Covington, my Dental Hygienist, is due a large part of the credit for arranging and preparing these charts.

DISCUSSION

Dr. Ketcham.—I am familiar to a greater or less degree with the good work which Dr. Hoffman is doing. He has had a class of dental hygienists in Denver whom he has trained in dietetics so that they can help the children or the children's mothers to select a well balanced ration, one that will aid these children in developing normally. It is something that we all should present to the mothers of our little patients. We can explain to them that if instead of choosing candy for the child, they would choose figs, dates, raisins, or ripe fruits, foods containing Nature's own sugar, the child will not crave so much manufactured sugar. Then explain to them how much better a food whole wheat is than the bolted flour, and the value of the leafy vegetables, rich in important vitamins, for salads.

The diet charts of Hoffman are most excellent. I wish we all might have a supply to give parents; then in spending more time in educating our clientele we will not only do a wonderful amount of good, but we will also lighten our own labors in treating malocclusion, for we will secure better health for our patients, and better development, with special reference to development of the bony structures. I am delighted that Hoffman has called this to the attention of our Society, and I know that experience along this line will make you all enthusiastic over its possibilities.

Dr. L. H. Wirt, South Bend, Ind.—There are many other men here who are much more representative than I am to discuss this paper, but there is one phase of this subject that Hoffman did not touch very seriously and that appealed to me as being important. I believe that the food habits and the habits of eating are formed some time earlier than would appear from the work that Hoffman has just shown us. I believe that the food habits of a child are really begun at about six or eight months.

I want to call attention to one thing which he only touched upon lightly, and that is in the matter of chewing the food. Doubtless, you have all had some experience that has brought you into contact with this question; if you have raised any children of your own, perhaps you have had this brought home to you. When the child is old enough to begin to take partially solid food, the first thing recommended by the physician is food which has to be mashed up or strained. I believe that it is during this period that the child learns to bolt the food, and after a habit is once formed it is very difficult to eradicate. I do not believe there is any other animal for which it is necessary to mash and strain food during infancy. and I see no good reason why a human infant should have that done either.

Now, if you will consider the function of swallowing, the tongue and the nervous connections of the tongue are so arranged that when the food has been taken into the mouth and acted upon by the teeth and jaws for a certain amount of time, it continues to pass further back until it gets to the back part of the tongue and by that time it is supposed to go down the throat. When it reaches a certain point in the throat, it comes in contact with the nerve endings which you might compare to an electric button and after that it cannot be stopped. If we take food into the mouth in a mashed condition, it goes back there with much less chewing and the child does not get a chance to exercise the instincts which the Lord gave him and he bolts his food from the very beginning. After the child has become a year to three years old, or a time when you can tell him he can have these things and not something else, and he is old enough to have this, that is too late. I believe you have to begin earlier. If you give the child proper instructions as soon as he begins to take solid food, I can see no harm in giving him raw fruits and vegetables of certain types in the form that he can take and chew on and he will not bolt them and swallow them or choke on them.

I have found in my short experience that in a great many cases of deficiency of arch development, which, of course, entails a later malocclusion, a great deal of good can be done by getting the child to chew more and thus exercise the jaws and those arches will develop wonderfully. So if you teach a child the habit of chewing before he has had a chance to acquire the habit of bolting the food, I believe we will accomplish a great deal for almost every child.

CALCIUM METABOLISM IN ITS RELATION TO DENTAL PATHOLOGY CALCIPRIVAL ODONTOPATHIA*

BY EDWARD L. MILOSLAVICH, M.D., MILWAUKEE, WIS.

(Professor of Pathology at the Marquette University, formerly Associate Professor of Pathological Anatomy at the University of Vienna, Austria.)

WHEN I accepted the invitation to address the Twenty-Third Annual Meeting of the American Society of Orthodontists on "Calcium Metabolism in Its Relation to Dental Pathology," I was fully aware of the task which confronted me.

The many dubious and obscure phenomena and factors as well as the numerous contradictory theories and conceptions which attend this problem render a discussion very difficult and even make a definite conclusion impossible.

This topic has been investigated by many prominent scientists from various viewpoints. I refer principally to MacCallum, as the representative of this country, and Erdheim, as the well-known investigator of the Vienna School, of which I am also a product.

Calcium metabolism is experiencing intensive chemical study and investigation at the present time. The most modern methods in metabolic research are being employed in a thorough and exhaustive manner in the attempt to solve this problem.

As a pathologist I will not treat the subject from the chemical aspect, as I lack the necessary experience in this particular field. I will, however, endeavor to discuss and to demonstrate morphologically the disturbances of calcium metabolism, to compare the human and animal pathology and to conciliate the experimental results with the clinical facts.

Consequently, my discussion will be limited and centered about the morphologic side of the question. In order to make the problem as clear as possible I will discuss the changes of the teeth and bones simultaneously. These two organs should not be treated separately in the consideration of this topic, for the reason that both are affected at the same time, by the same noxious agencies.

Basing my discussion on the experimental achievements of Erdheim, I will begin to describe the *calciprival changes of the teeth* (incisors and molars) of rats, as this particular animal is exceptionally well adapted for morphologic observations and consequent proof of disturbances in calcium metabolism.

Two structures of the tooth contain mainly calcium: dentine, the framework of the teeth, and cementum; but the structural alterations we will find particularly in the case of the former during the course of disturbed calcium metabolism.

Dentine, the chief constituent of the tooth, consisting of extremely fine

*Read at the Twenty-third Annual Meeting of the American Society of Orthodontists, on March 18, 1924, at Kansas City, Mo.

fibrillæ, which lie in close proximity to one another, embedded in a homogeneously calcified matrix, develops through appositional activity of the odontoblasts. A normal tooth never shows any irregularities in lime salts deposits in the dentine. In the tooth the calcification occurs in the so-called form of dentine globules, which are deposited very closely to each other.

The homogeneous distribution of the calcium in the dentine in the teeth of the rats gives the appearance of transparency to the normal tooth, while the lack of homogeneity in the calcium content, caused by a succession of calcified and noncalcified layers of dentine, produces the opaque appearance of the teeth. This phenomenon may be readily explained optically in that a medium with irregular structure possesses an unequal refractive index, which destroys the transparency of such a body.

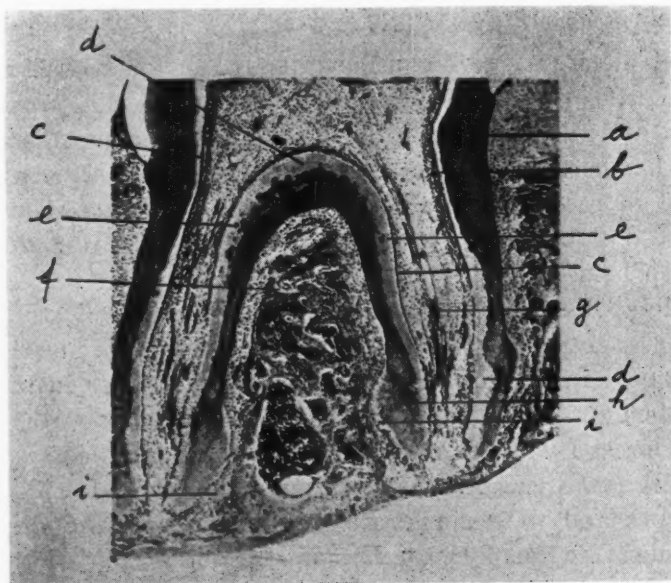


Fig. 1.—Root section of a rachitic molar tooth of a rat, 35 times enlarged (after Erdheim). The dentinoid area (*d*) of the root is enlarged and contains dentinal globules (*e*) which are either isolated or confluent, showing an irregular boundary toward the calcified zone of dentine. *a*, Homogeneously calcified dentine of the crown, nearly of normal width, with a sharp boundary toward a very thin dentinoid (*b*); note the opposite behavior in the same root; *c*, odontoblasts, in several places interrupted; *f*, osteoid tissue in jawbone; *g*, pulp with blood vessels; *h*, calcified cementum forming a club-shaped appearance at the apices of the roots; *i*, uncalcified cementum.

In opaque teeth we can microscopically distinguish a band-like structure of the dentine, which indicates a deposition of calcium in irregularly successive layers at irregular intervals (Figs. 1 to 5). This finding is typical in rachitis and also occurs after the experimental removal of the parathyroids, both conditions evidently accompanied by a disturbance in the calcium metabolism. Similar changes, that is, the successive depositions of calcified and uncalcified areas, were described by Pommer and others in the human bone tissue, especially in osteomalacic and rachitic human skeletons. These findings in both related organs are therefore a characteristic and morphologically demonstrable proof of a disturbance in calcium metabolism.

In rachitis we notice that the osteoblasts produce an osteoid tissue, which,

however, calcifies only incompletely, or delayed, or not at all, because there is a disturbance in the calcium circulation of the organism, which prevents a normal deposit of calcium salts in the developed and developing osteoid.

Identical conditions are encountered in the tooth. The odontoblasts first produce the uncalcified stage of the dentine, the so-called dentinoid, which shows a deficiency or irregularity in calcification, or a complete lack of lime salts.

Similarly parathyroidectomy prevents a partial or complete calcium saturation of the newly apposed dentine layers and causes a friability of the tooth, which may result in spontaneous fractures after seven to eight weeks (Erdheim, Fleischmann, Iselin, Leischner, Minkiewitsch).

Fleischmann, induced by the investigations of Erdheim on rats, was the first one to make accurate morphologic studies of the teeth of rachitic children. He found that the dentine in the vicinity of the pulp cavity presented a wide uncalcified zone, the identical finding seen in the case of rachitic rats.

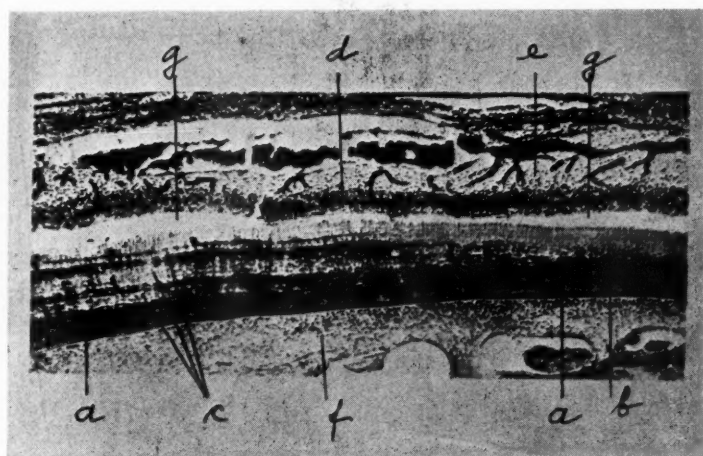


Fig. 2.—Mandibular rachitic incisor of a rat, 35 times enlarged, opaque in appearance (after Erdheim). *a*, Well calcified and *b*, uncalcified dentine, between both layers isolated dentinal globules are perceivable; *c*, large bands of uncalcified dentine (band-like arrangement of dentine) with inclusion of isolated and confluent dentinal globules; *d*, odontoblastic layer which is detached (*g*) artificially from dentine; *e*, pulp; *f*, alveolar epithelium.

Fleischmann even claims that rachitis in children may be more readily recognized by an examination of the teeth than of the bone.

It is interesting to mention that the deficiency or lack of calcification of the dentine can be demonstrated and studied by Roentgen rays (Erdheim, Toyofuku).

In skeleton diseases, which are caused by a disturbance in calcium metabolism, we may find bones which are impregnated uniformly but not completely with calcium salts; they show a *deficiency in calcium saturation*. Analogous conditions were found in teeth of rachitic rats and after the extirpation of parathyroids. The dentine showed a homogeneous and uniform impregnation with lime salts, but the deposit was not complete and did not reach the normal degree of saturation.

A further typical occurrence in the dentine during disturbed calcium metabolism is the *appearance of blood capillaries in the dentine substance*.

These capillaries penetrate and follow the direction of dentinal tubules, producing consecutive disturbances in the calcification (Fig. 4). In spontaneous rachitis, and after removal of the parathyroids, dentine remains uncalcified near the penetrated blood capillaries. It is rather significant that in human rachitis and osteomalacia identical changes occur in the osseous system; the lime salts are not deposited in the immediate neighborhood of the blood vessels; pure osteoid tissue surrounds them (Rindfleisch, Pommer).

Furthermore, Erdheim was able to demonstrate convincingly in the course of his studies on parathyreoprival changes of the dentine, that *the immediate vicinity of the dentinal tubules is the place of predilection for calcium deposit*.

We again observe the same phenomenon in rachitic and osteomalacic human bones, namely, that the deposit of lime salts takes place in close proximity to the bone canaliculi.

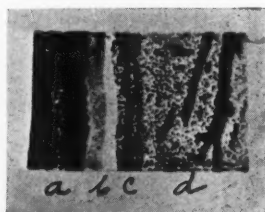


Fig. 3.—Section from Fig. 2, 35 times enlarged, showing details in the structure of dentine. *a*, Globular structure of dentine consisting of dentinal globules and uncalcified interglobular spaces (globular calcification); *b*, uncalcified dentine; *c*, odontoblastic layers; *d*, pulp with blood vessels.

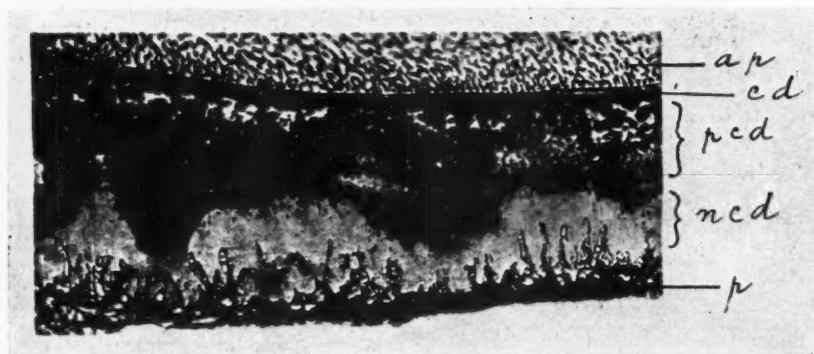


Fig. 4.—Incisor tooth of a rat, 101 times enlarged, 108 days after parathyroidectomy (after Toyofuku). Normally calcified dentine (*cd*) is represented by a small and thin layer; the partially calcified area (*pcd*) appears very large, containing in the outer portion distinct dentinal globules (globular calcification), which area is followed by a band-like arrangement of dentine (irregular calcification), its internal border being deeply indented. One notices numerous blood vessels growing out from the pulp (*p*) into the non-calcified dentine (*ncd*) producing a wavy boundary between the latter area and the partially calcified dentinal zone (spotted-like appearance). *ap*, Alveolar periosteum.

We, therefore, find that the dentine around the dentinal tubules shows an opposite behavior from that found in the presence of penetrated blood capillaries.

In these two morphologic findings we observe a complete agreement in the human and animal pathology.

To summarize, the *disturbance in the calcium metabolism expresses itself in the dentine* as follows: 1. band-like arrangement of the dentine (*irregular calcification*); 2. low degree of calcium saturation (*deficient calcification*);

3. vascularization of dentine (*spotted calcification*); 4. appearance of isolated dentine spheres (*globular calcification*).

These, with additional changes of the tooth still to be mentioned, occurring under similar conditions I should like to designate with the common term of "*calciprival odontopathia*."

The *dentinoid* represents a zone free from calcium, which lies between the layers of the odontoblasts and the dentine proper. The border of the dentinoid and the calcium containing dentine is normally linear or slightly wavy in molars as well as in incisors. The amount of the dentinoid under normal conditions may be considered very small, for it consists only of an extremely thin layer.

Only in very rare cases can dentine globules be found in normal teeth. These formations may be said to arise from irregular calcification of the dentinoid. They are typical findings in rachitic teeth (Figs. 2, 3).

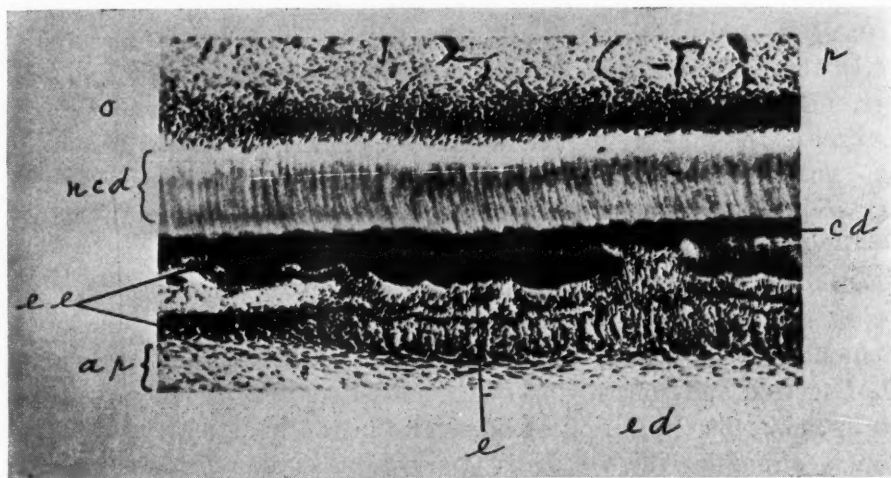


Fig. 5.—Incisor tooth of a rat, 80 times enlarged, 38 days after parathyroidectomy (after Toyofuku). The uncalcified dentine (*ncd*) is very large, while the normal calcified dentine (*cd*) shows just a thin and narrow line. The external border of the enamel (*e*) is wavy; the enamel presents a complete defect (corresponding to the projection of the letters *e d*), where the enamel epithelium (*ee*) is in direct contact with dentine (*cd*). The enamel epithelium (*ee*) also shows a wavy appearance corresponding to the external outline of enamel. At the left side of the picture one can notice a separation of the internal and external epithelial layers (*ee*). *p*, Pulp; *o*, odontoblasts; *ap*, alveolar periosteum.

Just as we notice a transitory calcium-free structure in the growth of the bone as the first tissue which later becomes impregnated with lime salts, we can distinguish the identical behavior in the development of the tooth. The dentinoid represents the transitory calcium-free tissue, the uncalcified stage of the dentine, which increases through appositional growth, thus imitating the growth of bone.

We can, therefore, expect that both organs, *tooth and bone*, react *simultaneously during disturbed calcium metabolism and subsequently represent similar or even identical morphologic changes and disturbances in growth*.

The rachitic changes in the growing tooth are furthermore characterized by an abnormal, often extreme, width and expansion of the dentinoid zone, which at times exceeds that of the dentine itself (Figs. 1 to 5). *The extent of the dentinoid zone is directly proportional to and depends upon the rachitic*

condition of the organism. In this disease we notice that the borderline between the dentinoid and the calcified dentine is never straight, but more or less irregular. This irregularity is partially caused by the above-mentioned penetration of blood capillaries from the pulp cavity into the dentine region.

Analogous conditions are to be observed in the skeleton of rachitic animals, where osteoid is being formed in great quantity and therefore shows the same general behavior as dentinoid. Both kinds of tissues represent primary stages of the firmest organs in the animal body and during the normal and pathologic growth act in the same manner.

The absence of calcium salts in the recently formed appositional area in the tooth as well as in the bone is a characteristic sign of a disturbance in calcium metabolism.

The *odontoblasts*, which line the surface of the pulp cavity, produce dentine, and between the two lies the dentinoid, the calcium-free phase of the dentine. The apposition of dentine proceeds slowly but steadily from the direction of the pulp, which is to be regarded as the nourishing tissue of the tooth. In this way the normal tooth is built in successive layers, which are, however, not distinguishable on account of the uniform and homogeneous distribution of the lime salts. After removal of parathyroids the odontoblasts undergo an atrophy, their cellular continuity becomes loose and broken (Fig. 1). The latter phenomenon can especially be seen in those places where blood vessels of the pulp cavity are penetrating into the dentinal tissue.

In the growth of teeth we would like to emphasize the *importance of local mechanical irritations*, which promote the growth by inciting and activating the odontoblastic cell tissue. The stimulus produced on the bone during the course of tension and contraction of the muscular groups not only favors its growth but also the deposition of lime salts in the bone matrix. Mastication produces a stimulus which evidently serves a similar physiologic purpose. Thus we notice for instance that the child while passing through the stage of dentition performs cramp-like movements of the jaws. We also observe the young animal, the puppy for example, vigorously and eagerly chewing hard objects. Both examples serve as an argument for the *physiologic necessity of an increased stimulus* and more intense activity, which are of great importance to the growth of the tooth and to the apposition of the lime salts.

The deficient development of *cementum* is often in direct proportion to the deficient growth of the skeleton. This is not surprising since, after all, cementum is nothing but bone tissue and therefore will be affected by the same agencies which are injuring the osseous system. In fact a parallelism can be observed between the amount of calcium in the cementum and skeleton. It is not altogether inconceivable that an increased resorption of cementum in advanced age, due to disturbed calcium metabolism, may be possible in man, resulting in a loosening of the teeth.

The pathologic changes in the *enamel* in rachitic rats (spontaneous and experimental rachitis), changes which are also found in the same animals after experimental removal of the parathyroids, again due to or followed by a disturbance in calcium metabolism, present a very interesting and significant problem. These changes are still more important in that they are analogous

to findings in human pathology, in teeth of tetanic children for example (Fleischmann, Spieler) which, as is well known, show disturbances in calcium metabolism. These changes are referred to as "hypoplastic enamel."

The enamel of the tetanic rats shows a marked reduction of the epithelial layers, even down to a single-celled layer, accompanied by pathologic processes in the epithelium of the enamel itself (adamantoblasts). This fact explains the *deficient formation of enamel*. As a matter of fact one can find instances where the enamel is completely absent or lacking, the so-called *enamel defect* (Fig. 5), and in this latter case one may observe a single row of a low enamel epithelium in close contact with the dentine. At times even the adamantoblasts may be completely absent. The injured enamel epithelium however makes efforts to further produce enamel, which may be found in the shape of structureless formations (like spheres, etc.) enclosed in the epithelium itself. The enamel eventually produced from the injured epithelium differs from the normal in the nature of the prismatic structure formed.

Thus we meet with disturbances in the growth of enamel epithelium, caused by a noxious agent, or through insufficient function of parathyroid glands.

Macroscopically a grooved and bandlike surface of the enamel, or a white spotted appearance may be observed.

The *morphologic changes in the enamel* of the growing tooth in parathyreoprival conditions as well as in disturbed calcium metabolism are: (1) hypoplasia of enamel; (2) defects in enamel; (3) reduction of the enamel epithelium, and (4) frustrated enamel formations within the epithelium itself.

These changes, principally those of the dentine, without a doubt bring about a decreased consistence and physical hardness of the tooth.

The most marked changes in disturbed calcium balance in the growing human organism are usually seen in the incisors. This fact we will try to explain with comparisons and findings from the pathology of the osseous system. In the study of bone calcification in cases of rachitis one may find that the greatest amount of lime salts is deposited in those parts most used, whereas, the greatest amount of osteoid tissue can be seen in those places least employed. The same *rule of calcium deposition* is to be applied to the tooth. The human incisors are the teeth which are used the least and therefore show the most pronounced disturbances; the molars on the contrary, perform most of the work in the act of mastication and therefore possess a heavier lime salt deposit. Similarly in the case of rachitic rats, the teeth most exercised show a better saturation with lime salts.

From the above discussion we may conclude that the same *static laws*, which regulate the deposit and distribution of calcium in the bones may be strictly applied in the pathology of the tooth.

The organism suffering from a deficiency in lime salts tends to dispose of even the small and insufficient amount of calcium, which may be present, to places where it is needed most urgently and imperatively. I should like to call this phenomenon: physiologic demand for skeletal hardness.

Allow me again to stress those factors in a different way, which I repeatedly emphasized, so as to keep them well in mind.

We are aware of one *fundamental factor regarding* the skeletal system: that apposition and resorption of the bone continues and persists throughout the life of the individual in the developing as well as in the fully developed bone; a slow but continuous transformation of bone tissue occurs throughout the entire lifetime of the human or animal organism.

Bone grows first by the apposition of a calcium free tissue known as osteoid, which under normal conditions becomes impregnated with lime salts till it finally calcifies completely and uniformly. *The parathyroids are those glandular organs which prepare and influence those tissues, which are potentially capable of receiving lime salts, like osteoid and dentinoid*; the parathyroids, in other words, condition these tissues for the reception of lime salts. In skeletal diseases, which are accompanied with disturbances in calcium metabolism, as found in the osteomalacic and rachitic group, the newly apposed calcium-free tissue does not exhibit any tendency or capability of attracting lime salts and it therefore remains devoid of lime salts. The parathyroids of such individuals exhibiting such diseases present pathologic changes.

The same rules are to be applied to the growing tooth, the adult tooth excepted, for the latter is a finished product, which is apparently no longer undergoing further structural metamorphosis. Deficient appositions during disturbed calcium metabolism, leaving indelible traces on the teeth of the growing organism, only occur during the developing period of the tooth.

The parathyroidectomy causes a profound alteration in the calcium metabolism, which can be traced both, chemically and morphologically. Chemically we find, that the calcium amount in the body, tissues and blood is considerably diminished, that the elimination of the lime salts increases: the body is gradually deprived of calcium salts (parathyreoprival calcium deficiency) (MacCallum and Voegtlin, Leopold, v. Reuss). Morphologically we consistently find the changes already mentioned in the skeleton, which are identical with the changes found in rachitic and osteomalacic individuals.

I do not want to dwell on the well-known experimental proofs regarding the influence which parathyroid feeding may have on the growth of bone and the formation of callus. Neither do I intend to discuss the pathogenesis of tetany, as you are familiar with this symptom complex.

The ingenious and conclusive experiments of Erdheim with the *transplantation of parathyroids* have proved that the calcification of dentine is influenced by and depends upon the presence of functional parathyroid tissue. After removal of the parathyroids a disturbance in the calcium balance takes place in the organism and the dentine which grows during this period does not assimilate any lime salts (rachitic reaction). If, however, parathyroids are transplanted so that they are able to function, a cessation in the disturbance of calcium metabolism takes place and the dentine, which is apposed immediately afterwards, is properly impregnated with lime salts (healing reaction). In such an experiment we notice the following gradation in the structure of the dentine:

1. Normally calcified dentine (preoperative stage);
2. Dentine devoid of

calcium (postoperative stage parathyroidectomy), and 3. Normally calcified dentine, so-called transplantation line (parathyroid transplantation).

This "transplantation line" is a convincing argument for the physiologic significance and effect of the parathyroid glands on the calcium metabolism.

I will now give a short résumé of such diseases of the human skeleton which are undoubtedly caused or accompanied by a disturbance in calcium metabolism and by more or less pronounced morphologic changes of parathyroid glands.

Pathologic anatomic changes of the parathyroid glands (processes of hyperplastic and proliferative character) were observed in:

Osteomalacia, Bauer, Erdheim, Hohlbaum, Schmorl, Todyo.

Puerperal osteomalacia, Strada.

Senile osteomalacia, Erdheim, Strada, Todyo.

Osteitis deformans, Molineus, Schmorl, Todyo.

Senile osteoporosis, Todyo.

Rachitis tarda, Erdheim.

Infantile rachitis, Erdheim, Hecker.

I feel it is necessary to mention that the parathyroids in rachitis and osteomalacia, which really are identical morphologic diseases, exhibit the same structural changes. The gradual transitions which have been described as occurring between fibrous osteitis and osteomalacia further indicate that the previously mentioned series of skeletal diseases belongs to a common, related group and exhibit a common pathogenic symptom.

Both, a low calcium diet and artificial injury to the calcium circulation, have been tried to produce in the skeletal system the same changes, which several authors (Dibbelt) explained as a true rachitis. Opposed to this view, however, other investigators (Goetting, Lehnerdt, Schmorl, Stoeltzner) claimed that an insufficient supply of lime salts in the organism cannot produce a true rachitic disease of the bone, but only the so-called "pseudorachitic osteoporosis," a somewhat different morphologic picture. We may truly then call the latter diseases "*alimentary osteopathies*." The artificially produced deficiency of lime salts can therefore not solely and of its own accord cause these changes.

Hence it is obvious that the parathyroids bear an essential relation to calcium metabolism and therefore to certain disturbances of growth of the bone and tooth. The question arises of what nature is this essential relation. *Do the parathyroids regulate the calcium metabolism, do they control it and dominate it, like the islands of Langerhans the carbohydrate metabolism, or do they simply react towards calcium metabolism in the same manner as interrenal tissue react towards cholesterol metabolism?*

Erdheim sees a certain parallelism between the structural changes and enlargements of the parathyroids and the changes of the hypophysis during pregnancy. Both organs react and enlarge on account of an increased functional demand; evidently these changes are therefore of a secondary nature.

We know, however, that primary injuries or diseases of the parathyroid glands, like removal or destruction of the same, etc., produce rachitic changes in the growing tooth and bone, which expresses an insufficiency in para-

thyroid activity. *Is the rachitic diseased skeleton not an expression of a primary insufficiency of the parathyroid glands, which secundarily must react with a compensatory hyperplasia of their inferior tissue?*

We may, I believe, compare the reactions of the hypophysis and parathyroids. The hypophysis enlarges during pregnancy and sometimes produces an acromegaloid condition; in other cases the enlargement of the hypophysis takes place without any perceivable symptoms.

It sometimes happens during pregnancy that the parathyroids become enlarged and this hyperplasia may occur without symptoms or it may be accompanied by a puerperal osteomalacia.

We could mention additional similar examples of abnormal metabolic reactions. *We observe the pathologic reactions in individuals of hypoplastic constitution who possess an inherited functionally deficient endocrine system, which fails during increased physiologic demand and secundarily presents morphologic changes of its own tissue.*

The same noxious agent which produces the calciprival odontopathia and osteopathia injures also the parathyroids which become insufficient and tend to offset this damage through a compensatory proliferation.

I fully realize that I have presented but a short sketch of a few salient facts, which comprise a meager part of the voluminous scientific data recorded about the question under discussion. I did not attempt to treat any detailed etiologic factors, since we are still uncertain about this phase of the subject, for the attainment of this desired goal is apparently far removed. I tried to merely mention a few pathogenetic moments, to demonstrate established morphologic facts and to enumerate our achievement and knowledge in this scientific field.

Further research may reveal new results to establish the fundamental principles of pathologic physiology of calcium metabolism.

My discussion should further illustrate that the investigation of an isolated organ is hardly sufficient to solve the facts completely. The examination of the entire human or animal body, the analysis of the related structures and organs enable us to comprehend the problem in its entirety. *Our concentration upon the diseased organ must not result in neglect of the whole diseased body, since the affected organ may only be a local manifestation of a systemic disease.*

If I have satisfied your expectations in the discussion of this topic I will consider my duty performed.

DISCUSSION

Dr. M. N. Federspiel, (Milwaukee, Wis.)—When the President said “a remarkable paper” I don’t think that quite covered it. It seems to me that when this paper is published and is read, so much of it will open our eyes to the realization that to study the tooth substance in itself is not sufficient, but that we should study the general anatomy and its relation to the parathyroids.

Dr. Miloslavich, for seventeen years a recognized authority in European countries in morphologic studies, comes to America, and we recognize him as a master pathologist.

We have heard him say that disturbance in calcium metabolism can be seen morphologically, and can be controlled under the microscope; that the changes of the teeth are

only symptoms of a systemic disease and, therefore, our attention should be directed towards both.

The detailed description of the different tissues of the tooth has shown us what wonderful reactions occur in a damaged organ. He goes on to tell us first there is a band-like arrangement of the dentine produced by irregular calcification. He tells us furthermore about different degrees of calcium saturation, and then about vascularization of dentine. He speaks of that as spotted calcification. He speaks of the appearance of isolated dentine pearls, and then sums it all up and defines it "calciprival odontopathia." That is something for the chairman of the Terminology Committee to remember.

This subject refers, to course, entirely to the tooth substance itself, but when we speak of the osteoid tissues involved in the jaw bone, I have just been wondering if there should be a deficiency of calcium in the jaws when we begin to correct the malocclusion of the teeth, and the teeth are placed in a normal occlusion and held so with retention appliances, how utterly impossible it is to keep the teeth in a normal position after the retaining appliances are removed. Therefore, gentlemen we have to go a little bit further and think of the function of the parathyroids in connection with postoperative appliances.

Dr. Waugh.—Mr. President and Fellow Members: I have enjoyed the paper immensely. It has introduced illuminating explanation of some of the things that have seemed obscure regarding the quality of tooth structure. It is a paper that must be carefully studied before one can form any accurate opinion of the influences upon the changes in developing tooth tissues which have seemed obscure. It is not a paper which one can discuss without having had the privilege of studying it, and even more than that, perhaps not without having had opportunity of conducting research along similar lines. So I feel that all I shall like to do at this time will be to express my appreciation of a very valuable contribution to our literature, something that will explain many obscure things and open up a field for much new thought. I personally am very grateful for the presentation.

Dr. Dewey.—I enjoyed the paper very much and the evidence which the Doctor has presented is quite conclusive along certain lines. However, if you have followed the literature the last few years in regard to the study of rickets, you will find that the profession may be divided into different groups. The essayist, of course, in his presentation called attention to the fact that there was a discrepancy among some pathologists as to what they considered a true rachitic condition. In certain diseases there is a faulty calcification which is produced by change in diet and would not necessarily be caused primarily by the parathyroids. I mention this to lead up to the fact that a peculiar thing exists in the study of calcific diseases such as rickets, because pediatricians admit that what they know as rickets can be treated successfully from three different sources. You probably know, some men claim that cod-liver oil and phosphorus produce beneficial effects in rickets in children; others claim the application of sunshine will produce equally wonderful results; then you will find a third group who claim the application of the quartz light to ricket individuals will have a beneficial effect. To that may be added the fourth group which claims rickets is caused by a disease of the parathyroid and a fifth who insist upon diet.

When you try to harmonize those various clinical observations and treatment of a disease which seem to have similar characteristics, you are somewhat at a loss to know how to proceed. But probably if you stop and remember that faulty calcification is a cell metabolic change, you may find those plans of treatment may not be so far separated. We find the study of endocrinology helpful in that there is a certain group of conditions certain pathologic changes that are not all the result of the disease of one particular gland but maybe of several glands. That was proved by the essayist when he referred to the changes that occurred to the parathyroid and hypophysis during pregnancy. Therefore, it is possible such agents as diet, sunshine, cod-liver oil and phosphorus and the result of the quartz light may produce cell metabolic changes that have an influence on the endocrine system.

So I think we must be careful in deciding or saying that calcific disturbances may

be the result of any one thing as long as clinical experience shows that result can be obtained in rachitic children by such widely different therapeutic agencies.

Dr. Ferris.—I have been greatly edified by the Professor's exhibition of his tremendous amount of work. The only thing I can say at this point which might be of value to us is the fact that we in our work make a direct stimulation, in Class II or Class III cases, in the movement of the mandible upon the parathyroid and the thyroid gland. We all know from our clinical observation without any special systematic recording that our children immediately improve in their general muscle tone. If we study the blood we find that the hemoglobin and the chemical balance between the calcium and phosphorous is greatly improved. So little work has been done in the chemical field that we are unable to make any definite statements, but our clinical observations are known to us all and we cannot help but feel a degree of satisfaction that our undertakings on the human body are resulting in constitutional value which may influence one set of the endocrine glands and that they may in turn influence all of the other endocrine glands in general.

I think that we have a rare treat in the presentation of this vital and scientific subject before this body.

Dr. Ketcham.—I feel that I can add but little to what has already been said.

In Dr. Dewey's summing up of the treatment of rickets, he neglected to add green vegetables, dairy products, etc., to the diet list. Then, too, I think it has been proved that sunshine has great virtue in the treatment of these conditions, but its virtues are almost nil under conditions of interference through window pane, screen, or soot in the air. Also, the use of the quartz lamp, as the Doctor mentioned, and ultraviolet rays, though not as good as direct sunlight in high altitudes or in a clear atmosphere, under conditions unfavorable for the use of direct sunlight, have their virtues in treating these conditions.

Dr. Howard.—Endocrinology has been a subject of considerable interest to me for the past several years. The Doctor's paper is most interesting. However, using as a basis the fact that interrelationship and interinfluence of glands exist, I would like to ask the Doctor if he is satisfied that the parathyroids *per se* are responsible for influencing calcium metabolism.

Observation has demonstrated to my mind and the minds of twenty-two other men at the Good Samaritan Clinic in Atlanta, that in cases of glandular dysjunction we often find a correlation of glandular insufficiency. In these cases there is often a failure of epiphyseal unions of the joints well into adult life.

It may also follow, I grant the essayist, that through that medium of imbalance the parathyroids have been affected. Perhaps the hypophysis is also affected, as well as the thymus. Just what is responsible for the failure of calcium metabolism and just which particular gland is responsible for that we do not know.

Dr. Hess of New York, who is considered one of our leading authorities on rickets, states that rickets has two specifics, namely, sunlight and phosphorus.

President Waldron.—Cod-liver oil added.

Dr. Howard.—He incorporated it, Doctor, but phosphorous is assimilated by the patient through the vehicle cod-liver oil. How can we harmonize sunlight and phosphorous, each being a specific, and at the same time take into consideration the parathyroids?

Gentlemen.—I believe it is impractical to burden any one or set off glands with the responsibility of a leading rôle in metabolism unless we have combined with that hypothesis practical, physical signs to corroborate our argument.

Dr. Miloslavich.—In the beginning of my paper I emphasized that I would not discuss the chemistry of metabolism; I omitted further the details in etiology and pathogenic factors as we are still in obscurity and are unable to determine positively which agencies cause these disturbances.

You certainly remember that series of diseases, which was projected on the screen;

it included the osteomalacic-rachitic group, the osteitis deformans and some diseases appearing in advanced age. All these skeleton changes are characterized by a disturbed calcium metabolism and nearly every one accompanied by an enlargement of the parathyroid glands and no other endocrine gland. We know that the organs of the endocrine system are correlated, that one tissue functions with the support of others. But I should not like to discuss these problems; there are so many theories connected with them. I prefer to speak of things which I can see, which I can touch and show, and not to discuss hypotheses and theories in a fantastic way. The treatment of rachitis may be sunshine, it may be cod-liver oil, or many other different procedures based on different theories. If we have a problem and many theories, then we are certain that our problem is absolutely obscure; if there is only one theory, then we are happy that we have solved our problem.

The question was asked, for instance, why the thymus does not present changes, why the hypophysis does not react, or any other of the organs. It was on the diseased human body that the morphologic reactions of parathyroids were found. In subsequent animal experimentations, in removal of parathyroids from a very sensitive animal such as the rat, the very same changes of the skeleton and teeth were found as in human rachitis.

From the etiologic standpoint rachitis is still an unsolved problem. We know, however, that rachitis and osteomalacia are the same morphologic process; we are unable under the microscope to distinguish between the two. Why can't osteomalacia be treated with sunlight? It is morphologically the same disease. There must be an agent about which we do not know. To solve that problem it is the duty of further research.

If I would undertake to review the entire endocrinology involved in this topic, it would take me too far and, as I emphasized, would bring me further into a field where I must discuss and operate with hypothetical questions. I tried only to show you reliable and demonstrable morphologic facts.

I again want to express my deep appreciation and my sincere thanks for the honor you have given me in having me appear before this highly scientific body.

OSSEOUS DEVELOPMENT IN ENDOCRINE DISORDERS*

BY WM. ENGELBACH, M.D., AND ALPHONSE McMAHON, M.D., ST. LOUIS, MO.

THE striking variations of stature in the eunuch, the acromegalic, and the various types of infantilism have been of historical interest even to the layman. An illustration of this interest is the prize offered by the philanthropist for the intermarriage of giants in order to produce a larger and stronger race. These outspoken differences in size and local osseous proportions of the individual were among the first signs giving impetus to the scientist to study the hormonie effects of the internal secretions upon skeleton growth. C. v. Langer (1872) was probably the first to distinguish between pathologic and normal giants. To Pierre Marie (1888-89), Massalongo (1892), Sternberg (1895), Brissaud and Meige (1895), and Launois and Roy (1902-04) credit should be given for the earliest work relating the development of the osseous system in gigantism and acromegaly to the hypophysis. Tandler and Grosz (1907-10) in their studies of the skopzen directly associated the overgrowth of the long bones present in the early hypogonad states to castration.

These early investigations, besides indicating a general change in the osseous system present in various ductless gland disorders, were pointing the way to local osseous abnormalities in the growth and development of individual bones as related to special glandular function. Soon after the discovery of the x-ray, roentgenology was applied with more minutiae to determine in the living the development of the osseous nuclei and the closure of the epiphyseal ends, which clinical deduction had predicted would vary in the endocrine subjects from those of the normal. Hertoghe (1896), v. Wyss (1899-1900), Kasowitz (1902), Diederle (1906), Siegert (1910), and others were the first to demonstrate by this means the presence of retarded carpal development in juvenile hypothyroidism.

In a clinical material of over 2,000 endocrine observations,† the striking differences in the osseous development, general and local, led the writers to suspect that there might be some specific predilection of the various hormones of special ductless glands in their effect upon the growth and development of the individual types of bones; i.e., short, flat, or long bones. This led to the following studies, which were undertaken to determine: (1) the normal osseous development in the various ages from one to twenty-five, during the skeletal growth; (2) the variations from this normal development in the various endocrine disorders, viz., thyroidism, pituitarism, gonadism, pinealism, thymolymphatism, pluriglandularism, etc.; and (3) the possible specific char-

*Address of President of Association for the Study of Internal Secretions, San Francisco, June 23, 1923.

Address delivered at Annual Meeting of the Radiological Society of North America, at Rochester, Minn., December 5, 1923.

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†Clinical observations of this material were made with Dr. J. L. Tierney.

acter of the internal secretions of these various glands in their effect upon the individual growth of one set of bones, such as the flat, long, or short. If such specific osseous hormone effects are presented, or even if definite general osseous changes, recognizable by roentgenograms, do occur in some of these endocrine disorders, they would be of great value in the early diagnosis

Chart I
UPPER EXTREMITY (GRAY'S ANATOMY)

BONES	APPEARANCE OF OSSIFICATION CENTERS	UNION
Humerus 8 centers	Shaft: 5th wk. fetal life. Head: 1st yr. Greater tubercle: 3rd yr. Lesser tubercle: 5th yr. Capitellum: End of 2nd yr. Medial epicondyle: 5th yr. Lateral epicondyle: 13th to 14th yr.	Head and tuberosities: 6th yr. Upper epiphysis with body: 20th yr. Lower epiphysis with body: 18th yr.
Ulna 3 centers	Body: 8th wk. fetal life. Head (inferior extremity): 4th yr. Olecranon: 10th yr.	Upper epiphysis: 18th yr. Lower epiphysis: 20th yr.
Radius 3 centers	Body: 8th wk. fetal life. Lower epiphysis: 2nd yr. Upper epiphysis: 5th yr.	Upper epiphysis: 17th to 18th yr. Lower epiphysis: 20th yr.
Carpal bones 1 center for each	Capitate: 1st yr. Hamate: 1st yr. Triangular: 3rd yr. Lunate: 5th yr. Greater multangular: 5th yr. Navicular: 6th yr. Lesser multangular: 8th yr. Pisiform: 12th yr.	
Metacarpals 2 centers	Body: 8th wk. fetal life. Distal extremity: 3rd yr. Base of thumb metacarpal: 3rd yr.	20th yr.
Phalanges 2 centers	Body: 8th wk. fetal life. Proximal extremity:- 1. First row: 3rd to 4th yr. 2. Second and third rows: 4th to 5th yr.	18th to 20th yr.

of the disorders of the glands, as well as a specific measure of their function and the treatment effects of these dyscrasias. These studies apply only to the age of twenty-five, at which time the osseous development is normally completed.

In order to draw any convincing conclusions from these comparisons, it was first necessary to determine a normal for each age and then to agree upon the definition of a grouping of the various endocrine disorders, both of which

precepts are very difficult and probably cannot be established beyond criticism. Before accepting individuals as normals for the various ages, they were carefully examined clinically and roentgenologically. The clinical examination consisted of investigating the family history, particularly for endocrine and hereditary conditions, including blood examinations of the parents. A complete medical survey of each individual, including comparison for the height, weight and age with the normal (established by tables of Browning, Bowditch

Chart II LOWER EXTREMITY (GRAY'S ANATOMY)		
BONES	APPEARANCE OF OSSIFICATION CENTERS	UNION
Os coxae 8 centers	Primary:- 1. Ilium: 8th wk. fetal life. 2. Ischium: 3rd mo. 3. Pubis: 5th mo. Secondary:- 1. Crest of ilium) 2. Antero-inferior spine) 3. Tuberosity of ischium) - puberty. 4. Pubic symphysis) 5. Acetabulum)	Ilium) Ischium) 18th yr. Pubis) Secondary and primary centers: 20th to 25th yr.
Femur 5 centers	Body: 7th wk. fetal life. Head: End of 1st yr. Greater trochanter: 4th yr. Lesser trochanter: 13th to 14th yr. Lower epiphysis: 9th mo. fetal life. 2nd to 3rd yr. Complete at puberty.	All united after puberty in reverse order of their ap- pearance. Lower epiphysis at 20th yr.
Patella 1 center		
Tibia 3 centers	Body: 7th wk. fetal life. Upper epiphysis: 2nd yr. Lower epiphysis: 2nd yr.	Lower with body: 18th yr. Upper with body: 20th yr.
Fibula 3 centers	Body: 8th wk. fetal life. Lower epiphysis: 2nd yr. Upper epiphysis: 4th yr.	Lower with body: 20th yr. Upper with body: 25th yr.
Tarsus 7 centers	Calcaneus: 6th mo. fetal life. Talus: 7th mo. fetal life. Cuboid: 9th mo. fetal life. 3rd cuneiform: 1st yr. 1st cuneiform: 3rd yr. 2nd cuneiform and navicular: 4th yr. Body: 9th wk. fetal life.	18th to 20th yr.
Metatarsals 2 centers	Base of first metatarsal: 3rd yr. Heads of second, third, fourth, and fifth: 5th to 8th yr.	
Phalanges 2 centers	Body: 10th wk. fetal life. Base: 4th to 10th yr.	18th yr.

and Holt for infants and children and insurance tables for adults) was then made. Provided the individual was considered normal according to these qualifications (i.e., family history, personal history, physical examination, and standard tables) he was then x-rayed and the roentgenologic findings were compared with those for the various ages as given by Gray's Anatomy and the radiologic studies of Baetjer and Waters, Knox, and Rotch. Decided variations, with personal exceptions enumerated in the following charts, in the roentgenologic determinations of the osseous system from those already con-

sidered within the normal by these four authorities were excluded as possible normals. Normals were examined eight to ten days after birth, at six months, one year, eighteen months, two years, and for each year up to the age of twenty-five. A number of each age were examined until enough were found without physical or roentgenologic variations from the normal to establish a standard. This work presented a great many difficulties and required a long time in order to exclude the abnormals physically and then finally to deter-

Chart III NORMAL OSSIFICATION AND UNION OF EPIPHYSES FOR YEARLY AGES (GRAY'S ANATOMY)			
YEARS	APPEARANCE AND UNION OF CENTERS	YEARS	APPEARANCE AND UNION OF CENTERS
Birth	Shafts of long and short bones. Calcaneus, talus, cuboid, third cuneiform (tarsals).	10	Olecranon (ulna).
1	Head of humerus. Head and distal epiphysis of femur. Capitate and hamate (carpals).	12	Pisiform (carpal).
2	Radius, lower epiphysis. Tibia, lower epiphysis. Fibula, lower epiphysis. Patella.	13 to 14	Lateral epicondyle of humerus. Lesser trochanter of femur.
3	Greater tubercle of humerus. Triangular (carpal). Hoods of metacarpals. Bases of phalanges (hand), first row. First cuneiform (tarsal). Bases of metatarsals.	16	Upper epiphysis of ulna with body.
4	Head of ulna. Bases of phalanges (hand), second and third rows. Greater trochanter of femur. Upper epiphysis of fibula. Second cuneiform and navicular (tarsals). Bases of phalanges (foot) - 4th to 10th yr.	17	Upper epiphysis of radius with body.
5	Lesser tubercle of humerus. Medial epicondyle, humerus. Upper epiphysis of radius. Lunate and greater multangular (carpals). Hoods of metatarsals - 5th to 8th yr.	18	Centers of femur. Lower epiphysis of humerus with body. Bases and bodies of phalanges (hands and feet). Lower epiphysis of tibia with body. Hoods and bodies of metatarsals.
6	Navicular (carpal). Union of head of humerus with tubercles.	20	Upper epiphysis of humerus. Lower epiphysis of ulna. Lower epiphysis of radius. Hoods and bodies of metacarpals. Lower epiphysis of femur. Upper epiphysis of tibia. Lower epiphysis of fibula.
8	Lesser multangular (carpal).	25	Upper epiphysis of fibula.

mine the roentgenologic normal. The four authorities quoted above giving normals for the various ages differed considerably in their opinions upon certain osseous developments as displayed by the roentgenogram, as shown by Charts I to VII.

In order to obtain some estimation of the roentgenologic findings of the various ages, the individual bones of the body were first classified from an anatomic viewpoint, as given by Gray's Anatomy, Charts I and II, on pages 3 and 4.

The bones according to the appearance of centers of ossification and union of the epiphyses for the yearly ages were then *arranged by years* according to Gray's Anatomy (Chart III).

The appearance and union of the centers of various bones of the body were then compared, according to Gray's Anatomy and the roentgenographic studies of Baetjer and Waters and Knox (Charts IV and V).

This information was rearranged as to yearly appearance and union of the

Chart IV APPEARANCE AND UNION OF BONE CENTERS, UPPER EXTREMITY, ACCORDING TO					
BONES	GRAY'S ANATOMY		BAETJER AND WATERS		KNOX
	APPEAR	UNITE	APPEAR	UNITE	
Scapula					
1.Acromion (2 cent.)	15th-16th	25th	15th	18th	22nd-25th
2.Coracoid (" ")	1st-17th	"	1st	15th	15th
3.Inferior angle	16th	"	15th	18th	22nd-25th
Clavicle					
1.Sternal end	18th-20th	25th	15th-17th	23rd-25th	25th
Humerus					
1.Head	1st	Head & tub- erosities, 6th.	6th-7th mo.	6th	5th
2.Greater tubercle	3rd	Head & shaft,	3rd-4th yr.	"	"
3.Lesser	5th	20th.	"	"	Head and shaft, 20th.
4.Capitellum	2nd	4, 5, 6 & 7:	1st	18th-19th	16th-18th
5.Medial epicondyle	5th	Unite with	5th	"	"
6.Trochlea	12th	shaft at	10th-11th	"	"
7.Lateral epicondyle	13th-14th	18th yr.	12th-14th	"	"
Ulna					
1.Lower epiphysis	4th	20th	4th	18th	20th
2.Olecranon	10th	16th	8th-9th	17th	16th
Radius					
1.Lower epiphysis	2nd	20th	2nd	17th-18th	20th
2.Upper	5th	17th-18th	5th	16th-17th	17th-18th
Carpal Bones					
1.Capit. (os magnum)	1st		1, 2, 3, 4, 5, 6 & 7: Ap- pear in ana- tomical ord- er, one bone for each year.		
2.Hamate (unciform)	"				
3.Triang. (cuneiform)	3rd				
4.Lunate (semilunar)	5th				
5.Gr.mult. (trapezium)	"				
6.Navicular (scaphoid)	6th				
7.Less.mult. (trapez- oid)	8th				
8.Pisiform (pisiform)	12th		8. 8th-11th		
Metacarpals					
1.Digital extremity	3rd	20th	3rd	17th-18th	18th
2.Base of 1st	"	"	"	"	"
Phalanges					
1.Head, 1st row	3rd-4th	18th-20th	3rd	16th-17th	18th-20th
2. " 2nd and 3rd rows	4th-5th	"	"	" 4th-5th	"

centers of ossification, according to Gray's Anatomy, Baetjer and Waters, Knox, and Rotch (Charts VI and VII).

It will be noted that there are variations of two to four years in the roentgenographic studies of Baetjer and Waters, Knox, and Rotch and the dissection made by the anatomists. For instance the anatomist gives the union of the lower epiphysis of the femur at the age of 20, while Baetjer and Waters state that this epiphyseal line disappears at 18 to 20. The heads of the metacarpals are given by Gray's Anatomy as completely united at 20, by Baetjer and Waters at 17 to 18, by Knox at 18, and, according to our normals, in all instances at 14 to 15. Other variations will be noted in almost

Chart V
APPEARANCE AND UNION OF BONE CENTERS, LOWER EXTREMITY, ACCORDING TO

BONES	GRAY'S ANATOMY	BAETJER AND WATERS	APPEAR	UNITE	APPEAR	UNITE	APPEAR	UNITE
Os Coxae								
1. Prim. centers								
a. Ilium	Birth	18th	Birth	15th-16th	Birth	18th		
b. Ischium	"	"	"	"	"	"		
c. Pubis	"	P & I, 7th-8th	"	P & I, 7th-9th	"	P & I, 7th-8th		
2. Sec. centers								
a. Ant.-inf. sp.	Puberty	20th-25th	15th	20th	Puberty	25th		
b. Tub. ischium	"	"	"	"	"	"		
c. Pubic symph.	"	"	15th-18th	23rd-25th	"	"		
d. Crest ilium	"	"	15th-16th	15th-16th	"	"		
e. Acetabulum	"	"			"	"		
Femur								
1. Head	End of 1st	Reverse order	End of 1st	17th-18th	End of 1st	Reverse order		
2. Greater troch.	4th	of appearance,	4th	18th	4th	of appearance,		
3. Lesser "	13th-14th	after puberty.	11th-13th	17th	13th-14th	after puberty.		
4. Lower epiph.	9th mo. fet.	4. 20th	Birth	18th-20th	9th mo. fet.	4. 20th		
Patella	1 life				1 life			
	2nd-3rd		3rd-4th		3rd			
Tibia								
1. Upper epiph.	2nd	20th	1st	18th-20th	Birth	20th		
2. Lower "	"	18th	2nd	17th-18th	2nd	18th		
Fibula								
1. Upper epiph.	4th	25th	3rd-4th	18th-25th	4th	25th		
2. Lower "	2nd	20th	2nd	17th-18th	2nd	20th		
Tarsus								
1. Calcaneus (os calcis)	6th mo. fet. life	Cent. for epiphysis os calcis appears at 10th yr. and unites after puberty.	Ref. birth	Cent. for epiphysis os calcis appears at 10th yr. and unites at 18th.	6th mo. fet. life	Cent. for epiphysis os calcis appears at 10th yr. and unites after puberty.		
2. Talus (astragalus)	7th mo. fet. life		"		7th mo. fet. life			
3. Cuboid (os cuboideum)	9th mo. fet. life		"		9th mo. fet. life			
4. 3rd cuneiform (external)	1st		1st		1st			
5. 1st (internal)	3rd		3rd		3rd			
6. 2nd (middle)	4th		4th		4th			
7. Navicular (scaphoid)	"		"		"			
Metatarsals								
1. Base of 1st	3rd	18th-20th	3rd-7th	About 17th	3rd	18th-20th		
2. Heads of 2nd to 5th	5th-8th							
Phalanges								
1. Bases	4th-10th	18th	3rd-7th	17th	Not given			

Chart VI
APPEARANCE AND UNION OF BONE CENTERS BY YEARS, ACCORDING TO

Yrs.	GRAY'S ANATOMY	BAETJER AND WATERS	KNOX	ROTCH
1	Coracoid proc. scapula Head humerus Capitate & hamate Head femur (birth) 3rd cuneiform	Coracoid proc. scapula Head humerus (6-7 mos.) Capitellum, humerus Capitate & hamate Head femur Upp. epiph. tibia 3rd cuneiform	Coracoid proc. scapula Head humerus Capitate & hamate Head femur Upp. epiph. tibia 3rd cuneiform	Head humerus (6-8 mos.) Capitellum, humerus (2-3) Capitate & hamate Head femur Upp. epiph. tibia (birth)
2	Capitellum, humerus Low. epiph. radius Patella (2-3) Upp. epiph. tibia Low. epiph. tibia Low. epiph. fibula	Low. epiph. radius Low. epiph. tibia Low. epiph. fibula	Capitellum, humerus Low. epiph. radius Low. epiph. tibia Low. epiph. fibula	Low. epiph. radius (2-4) Patella (2-3) Low. epiph. tibia (12th mo.-2nd yr.) Low. epiph. fibula (2-3)
3	Gr. tubercle humerus Os triangularis Heads metacarpals Heads prox. phalang (3-4) 1st cuneiform Heads metatarsals (3-8)	Gr. tub. humerus (3-4) Os triangularis Heads metacarpals Heads phalanges Patella (3-4) 1st cuneiform Heads metatarsals (3-7)	Gr. tubercle humerus Os triangularis Heads metacarpals Heads prox. phalang (3-4) Patella 1st cuneiform Heads metatarsals	Tuberosities humerus (2-3) Os triangularis (2-3) Heads metacarpals Heads phalanges (3-4) 1st cuneiform Heads metatars. (3-8)
4	Low. epiph. ulna Heads phalanges, 2nd & 3rd rows Gr. troch. femur Upp. epiph. fibula 2nd cuneiform Navicular (tarsal)	Low. epiph. ulna Gr. troch. femur Upp. epiph. fibula (3-4) 2nd cuneiform Navicular (tarsal)	Low. epiph. ulna Heads 2nd & 3rd phalanges Gr. troch. femur Upp. epiph. fibula 2nd cuneiform Navicular (tarsal)	Styloid proc. ulna Lunate (4-5) Upp. epiph. fibula 2nd cuneiform Navicular (tarsal) Heads phalang. (feet) (4-9)
5	Med. epicond. humerus Upp. epiph. radius Lunate & gr. multang.	Med. epicond. humerus Upp. epiph. radius Lunate & gr. multang.	Med. epicond. humerus Union head & tubercles humerus Upp. epiph. radius Lunate & gr. multang.	Med. epicond. humerus U. head & tub. humerus Upp. epiph. radius Low. epiph. ulna (5-7) Gr. multangular Navic. (carpal) (5-6) Gr. troch. femur
6	Union head & tubercles humerus Navicular (carpal)	Union head & tubercles humerus Navicular (carpal)	Navicular (carpal)	Less. multang. (6-8) Union isch. & pub. (6-8)
7	Union isch. & pub. (7-8)	Less. multang. (7th?) Union isch. & pub. (7-9)	Less. multang. (7th?) Union isch. & pub. (7-8)	
8	Less. multangular	Olecranon, ulna (8-9) Pisiform (8-11)		Epiph. os calcis (9th)
10	Olecranon, ulna Epiph. os calcis	Trochlea, humerus (10-11) Epiph. os calcis	Olecranon, ulna Epiph. os calcis	Trochlea, humerus Olecranon, ulna
11	Less. troch. femur (11-13)	Less. troch. femur (11-13)		
12	Trochlea, humerus Pisiform	Lat. epicond. humerus (12-14)	Trochlea, humerus Pisiform	Lat. epicond. humerus (12-13) Pisiform Less. troch. femur (12-14)

every age. It occurred to us that possibly the anatomist, pediatrician (Rotch), and radiologists (Baetjer and Waters) did not consider the endocrine possibilities in their so-called normals. This might explain the marked variability in age of the appearance of the centers of ossification and the fusion of the epiphyseal lines, as well as the general variations in the growth of the various bones.

After carefully interpreting this work and comparing with the normal that we had observed for the various ages, we have finally established the table above (Chart VIII), which is a compilation of our own studies and those of the other authors. In this article in referring to the normal, we use

Chart VII
No. 2 APPEARANCE AND UNION OF BONE CENTERS BY YEARS, ACCORDING TO

Yrs.	GRAY'S ANATOMY	BAETJER AND WATERS	KNIX	ROTCH
13	Lat.epicond.humerus (13-14)		Lat.epicond.humerus (13-14) Less.troch.femur(13-14)	
15	A.of acromion (15-16) Sec.centers os coxae U.of epiph.os calcis	A.of acromion Inf.angle scapula U.centers scapula (15-18) Sternal end clav. (15-17) A.of sec.centers os coxae a.Crest ilium (15-18) b.Acetabulum (15-16) U.of prim.centers os coxae	A.of acromion (15-17) Inf.angle scapula Sec.centers os coxae U.of epiph.os calcis	Union of:- Epiph.os calcis Prim.centers os coxae
16	A.of inf.angle scapula U.of olecranon	Union of:- Upp.epiph.radius (16-17) Heads phalang.hand(16-17)	Union of:- Dist.extr.humer.(16-18) Olecranon	
17	Union of:- Upp.epiph.radius(17-18) Less.troch.femur	Union of:- Olecranon Low.epiph.radius (17-18) Heads metacarpals (17-18) Head femur (17-18) Less.troch.femur	Union of:- Less.troch.femur Upp.epiph.radius(17-18)	
18	A.of sternal end clavicle (18-20) Union of:- Dist.extr.humerus Heads phalang.hand (18-20) Prim.centers os coxae Head femur Gr.troch.femur Low.epiph.tibia Heads metatars.(18-20) Heads phalang.foot	Low.epiph.tibia (17-18) Low.epiph.fibula (17-18) Heads metatars.(about 17) Heads phalanges foot (about 17) Union of:- Dist.extr.humerus (18-19) Low.epiph.ulna Gr.troch.femur Low.epiph.femur (18-20) Upp.epiph.tibia (18-20) Upp.epiph.fibula (18-25)	A.of sternal end clavicle (18-20) Union of:- Heads metacarpals Heads phalang.hand (18-20) Prim.centers os coxae Head femur Gr.troch.femur Low.epiph.tibia Heads metatars.(18-20)	A.of sternal end clavicle Union of:- Gr.troch.femur Less.troch.femur
20	Union of:- Upp.epiph.humerus Low.epiph.ulna Low.epiph.radius Heads metacarpals Sec.centers os coxae (20-25) Low.epiph.femur Upp.epiph.tibia Low.epiph.fibula	U.of sec.centers os coxae	Union of:- Upp.epiph.humerus Low.epiph.ulna Low.epiph.radius Sec.centers os coxae (20-25) Low.epiph.femur Upp.epiph.tibia Low.epiph.fibula	
25	Union of:- Centers scapula Sternal end clavicle Upp.epiph.fibula	U.of sternal end clavicle (23-25)	Union of:- Centers scapula (22-25) Sternal end clavicle Upp.epiph.fibula	

the osseous developments as given in the above table (Chart VIII). In order to make this work practicable and applicable to the average x-ray technician, we have devised a table (Chart IX) giving the parts to be x-rayed in individuals of various ages.

On account of the marked variations for each age, it is unnecessary to refer to the fact that, unless roentgenograms are taken of special bones for an individual age, the most important evidence referable to the osseous development for that age will not be obtained. It has been our custom in these cases to take roentgenograms of the bones given under the years before and after the age of the patient. For instance, for a patient who is nearest the tenth birthday, roentgenograms of the special parts given under the ages of 9, 10 and 11 in Chart IX should be made for comparison with the normal for those ages.

COMPARISON OF NORMALS WITH ENDOCRINOPATHIC SUBJECTS OF THE SAME AGE

Birth to the age of one. The normal infant (Fig. 1) shows the centers for the distal epiphysis of the femur, proximal epiphysis of the tibia, and three tarsal bones, the talus, cuboid, and calcaneus. There is absence of all carpals and heads of metacarpals and phalanges. The presence at birth of

Chart VIII
APPEARANCE AND UNION OF BONE CENTERS
ENGELBACH AND MCMAHON

Yrs.		Yrs.	
1	Coracoid process scapula Head of humerus (6-7 mos.) Capitate and hamate Head of femur Upper epiphysis tibia (birth) Third cuneiform	13	Lesser trochanter femur
2	Greater tubercle humerus Capitellum, humerus Lower epiphysis radius Patella (2-3 yrs.) Lower epiphysis tibia Lower epiphysis fibula First and second cuneiforms (2-4 yrs.)	14	U. of heads of metacarpals. (14-16 yrs.)
3	Os triangularis Heads of metacarpals Heads of phalanges Heads of metatarsals (3-7 yrs.)	15	A. of acromion Inferior angle scapula U. of centers of scapula (15-18 yrs.) A. of sternal end clavicle (15-17 yrs.) U. of heads of phalanges, hand A. of secondary centers of coxae a. Crest of ilium (15-18 yrs.) b. Acetabulum (15-16 yrs.) U. of primary centers of coxae
4	Lunate Greater trochanter femur Upper epiphysis fibula (3-4 yrs.) Navicular (tarsal)	16	Union of:- Distal extremity humerus Olecranon, ulna Upper epiphysis radius Heads of metatarsals Heads of phalanges, feet
5-6	U. of head and tubercles humerus Medial epicondyle humerus Upper epiphysis radius Greater multangular Lesser multangular (6-8 yrs.) Navicular (carpal) (5-6 yrs.)	17	Union of:- Lower epiphysis radius Lesser trochanter femur
7	Lower epiphysis ulna U. of ischium and pubis Epiphysis of calcis (7-8) Pisiform (9-11) Olecranon, ulna	18	Union of:- Head of humerus Head of femur Greater trochanter femur Lower epiphysis tibia
9		18-20	Union of:- Lower epiphysis ulna Secondary centers of coxae (20-25 yrs.) Lower epiphysis femur Upper epiphysis tibia Lower epiphysis fibula Upper epiphysis fibula
10	Trochles, humerus	22-25	U. of sternal end clavicle
11	Lateral epicondyle humerus (11-12 yrs.)		

the center for the upper epiphysis of the tibia is not generally admitted by all authorities. At the age of one there are normally two carpal centers present, the capitate and hamate. The head of the humerus, head of the femur, and third cuneiform appear within the first year. The absence of the capitate and hamate is particularly valuable in the diagnosis of early hypothyroid states. The diagnosis can thus be made at a time when material assistance can be rendered the child in the progress of normal development. For

second year. Other important centers occurring within the second year are the greater tubercle of the humerus, capitellum, patella, lower epiphyses of the tibia and fibula, and first and second cuneiforms, these last two appearing from two to four years. No. 2 demonstrates the effect of the administration of thyroid substances upon the osseous development. At forty-one months there are four carpal bones present, together with the head of the radius. The carpal bones are slightly undeveloped, with the os triangularis and lunate of

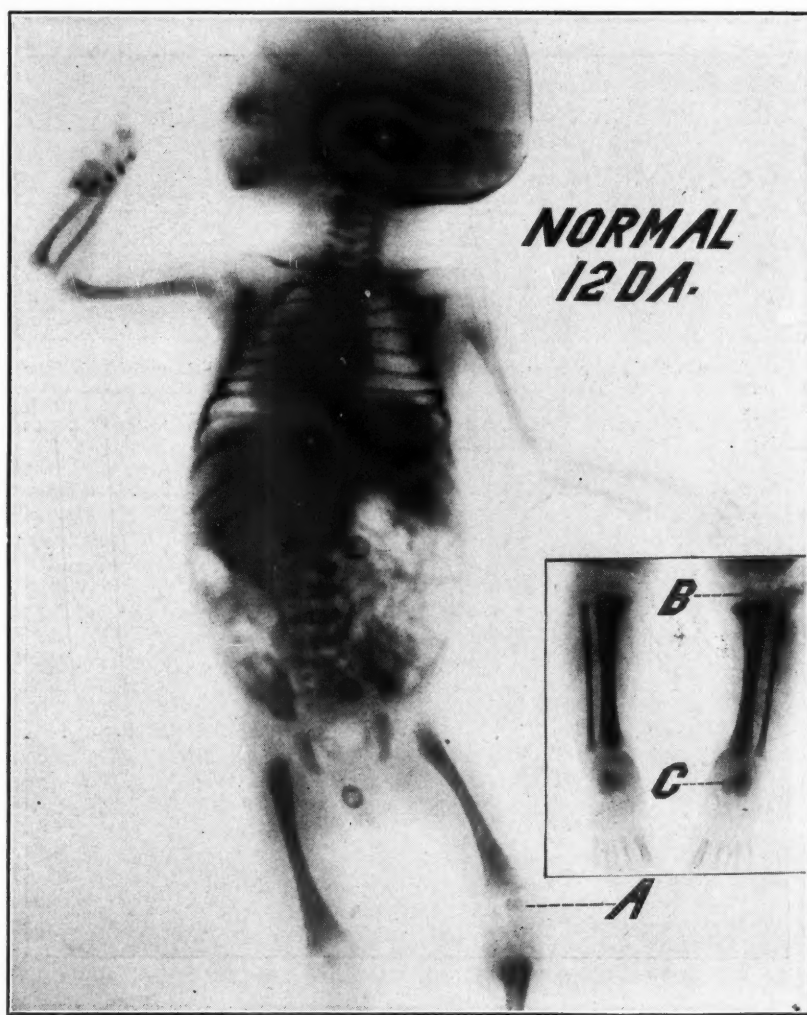


Fig. 1.

about equal size. No. 3 shows the same subject at fifty-six months. There has been definite progression in the development of the carpal bones and the head of the radius. The center for the head of the first metacarpal is also present. This hand is practically normal for the age.

Fig. 3 is a comparison of the same hypothyroid hand after treatment. The progress of the osseous development of the hand is noted in all epiphyseal centers, particularly in the heads of the metacarpals and phalanges. The previous figure shows the absence of many centers in the phalanges at forty-one months.

Aged two years, ten months. Fig. 4 is a demonstration of an enlarged thymus, with underdevelopment of the carpal bones and absence of the head of the radius. Normally at this age there should be three carpal bones present. The os triangularis is absent. Centers for some metacarpals are just appearing, which is within the normal. The heads of the phalanges are absent, these normally appearing within the third year.

Aged three. An example of a normal variation in the development of the carpal bones at this age is shown in Fig. 5. The picture to the left shows

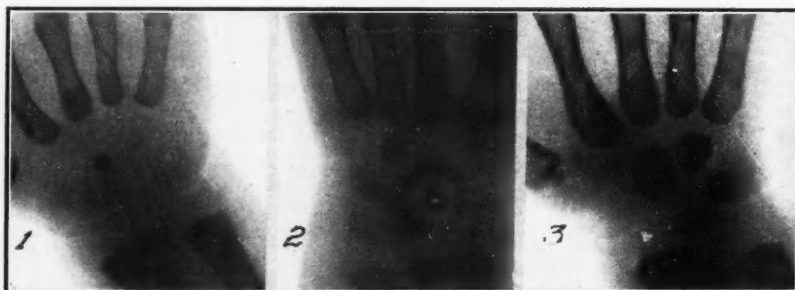


Fig. 2.—Effect of treatment upon hypothyroid. (1) 26 months old, before treatment. Note absence of hamate, os triangularis, and head of radius. (2) Same case after treatment, at 41 months. Note head of radius, capitate, hamate, os triangularis, and lunate. (3) Same case at 56 months. Note enlargement of head of radius and carpal bones present in Figure 2, and presence of head of first metacarpal.

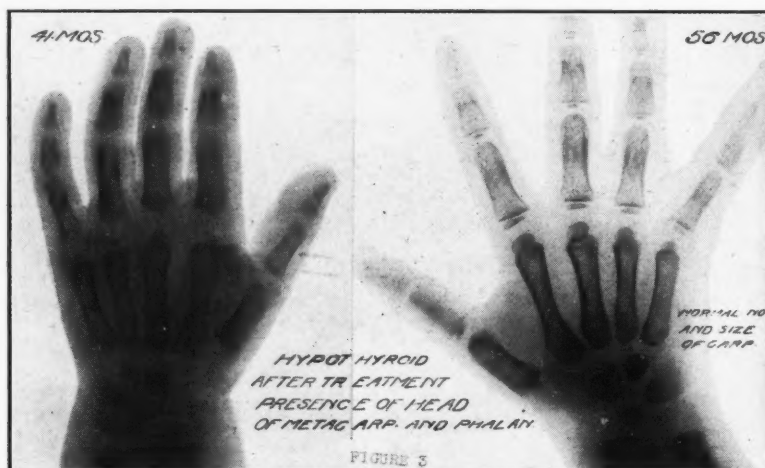


Fig. 3.

three carpal bones, the capitate, hamate, and os triangularis, with a well developed head of the radius. The one to the right shows four carpal bones, with appearance of the lunate and faint appearance of the head of the radius. The head of the first metacarpal is present on the left, and absent on the right. These variations occur in subjects who are of normal type. A slightly advanced development of the carpals, with apparently delayed development of the head of the radius, is not an infrequent association.

A comparison of the shoulders of a normal and a hypothyroid subject of three years is shown in Fig. 6. The normal shoulder on the left shows the greater tubercle well developed. This tubercle normally appears in the sec-

ond year. It is completely separated from the head of the humerus, establishing its separate origin. There has been some contention about this point. The hypothyroid shoulder on the right shows the head of the greater tubercle just appearing, consequently undeveloped representing a development of about nineteen months.

Aged four. Fig. 7 compares the normal with a hypothyroid hand of the same age. The normal hand shows four carpals present, the capitate, hamate, os triangularis, and os lunatum. The heads of all metacarpals and phalanges are well developed. The hypothyroid hand at four shows two carpals, the capitate and hamate, while the triangular and lunate are absent. The heads of the metacarpals and phalanges, with the exception of the heads of some phalanges, are present but undeveloped for the age. The carpal development is to the age of two. The delayed development of the carpals is an out-

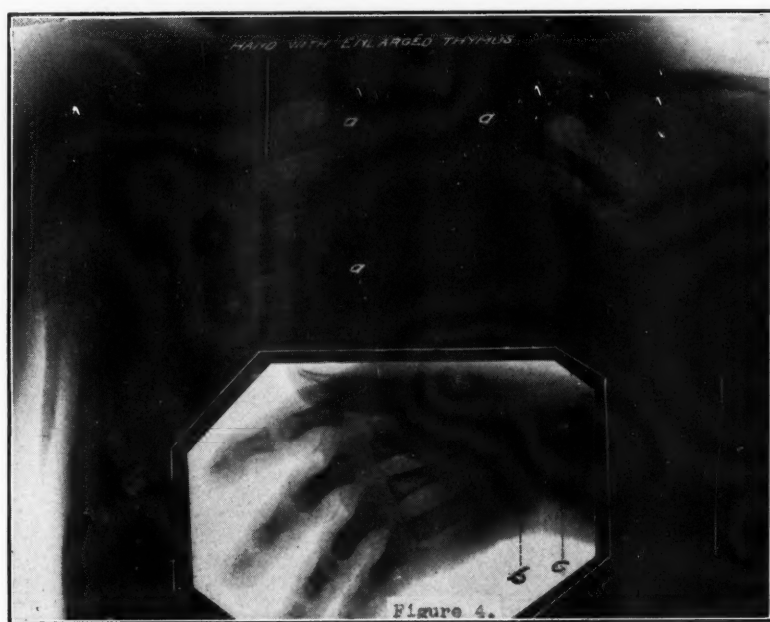


Fig. 4.—Aged 2 years, 10 months. Thorax: (a, a, a) Enlarged thymus. Insert of hand: (b) Absence of os triangularis. (c) Absence of head of radius. Note absence of heads of metacarpals.

standing feature in hypothyroid cases, although rather constant delayed development in the epiphyses of all long bones has been found.

A comparison of a normal with a hypothyroid shoulder at the age of four is made in Fig. 8. The greater tubercle of the humerus is normal in the picture on the left, with beginning fusion with the head. In the hypothyroid subject, the greater tubercle is undeveloped, with complete separation from the head, thus emphasizing the importance of centers other than the carpals.

A radiogram of the pelvis at the age of four (Fig. 9) demonstrates the center for the greatest trochanter of the femur (A) present in the normal subject (No. 1). This center (A) is absent in No. 2. There is also a difference in the development of the head of the femur. It is to be noted that the separation of the pubis and ischium is more pronounced in the hypothyroid pelvis (No. 2). These do not normally fuse until about the seventh year.

The radiograms of the knee joint at the age of four in Fig. 10 show in the normal subject on the left the presence of a fairly well developed upper epiphysis of the fibula, which normally appears between the third and fourth years. This center is absent in the hypothyroid knee on the right, and there is general underdevelopment of the other centers present, i.e., the lower epiphysis of the femur and the upper epiphysis of the tibia. This further emphasizes the point made above, that the underdevelopment of the osseous system in hypothyroidism is not limited to the carpal bones.

Fig. 11 illustrates the development of the feet at the age of four. The normal foot on the left shows the appearance of the center for the navicular, with the other tarsals well developed, the cuboid and the first, second, and

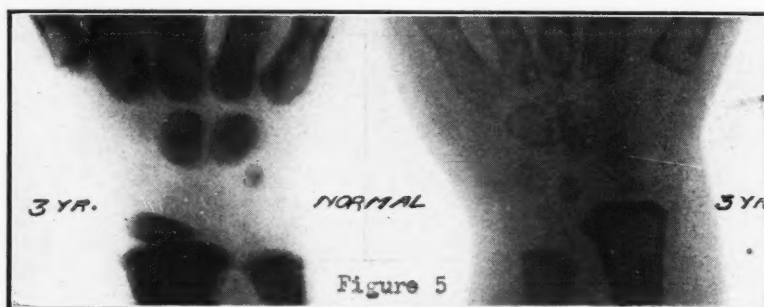


Fig. 5.—Carpal bones for normal, aged 3. Showing normal variation in appearance of carpals. Note well developed head of radius on left, and appearance of head on right. Also presence of head of first metacarpal on left, absence on right.

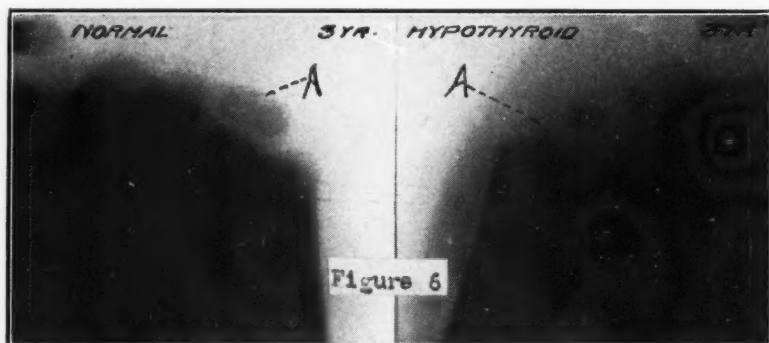


Fig. 6.—(1) Normal, aged 3. (A) Greater tubercle of humerus well developed. (2) Hypothyroid, aged 3. (A) Greater tubercle of humerus undeveloped.

third cuneiform. These last three appear within the first two years. The illustration on the right, a hypothyroid foot, shows the navicular and second cuneiform missing. The center for the first cuneiform, appearing within the second year, is faintly seen. It is to be noted that the center for the head of the first metatarsal is also undeveloped, as contrasted with the normal foot. The hypothyroid foot represents about a two-year development.

Fig. 12 is a radiogram of the carpal bones of a subject aged four with a clinical diagnosis of hypothyroidism. It is interesting to note that there is an overdevelopment of the carpal bones, the six present being the capitate, hamate, os triangularis, lunate, navicular, and lesser multangular. There is also an accessory head of the second metacarpal. The hand represents about

a six-year development. It is in these cases that the study of the osseous development assumes importance for diagnostic purposes. We assume a pluriglandular involvement, which may account for the advanced carpal development, not typical of the true cretin.

In Fig. 13 the hand of a normal four-year child is contrasted with that of a subject with suspected hyperpinealism. The normal hand is smaller than the comparison hand, while the carpal bones are present for the age. The suspected hyperpineal hand is developed to the age of twelve, with all the carpals present, including the pisiform. The heads of the metacarpals and phalanges are likewise well developed, together with the heads of the radius and ulna

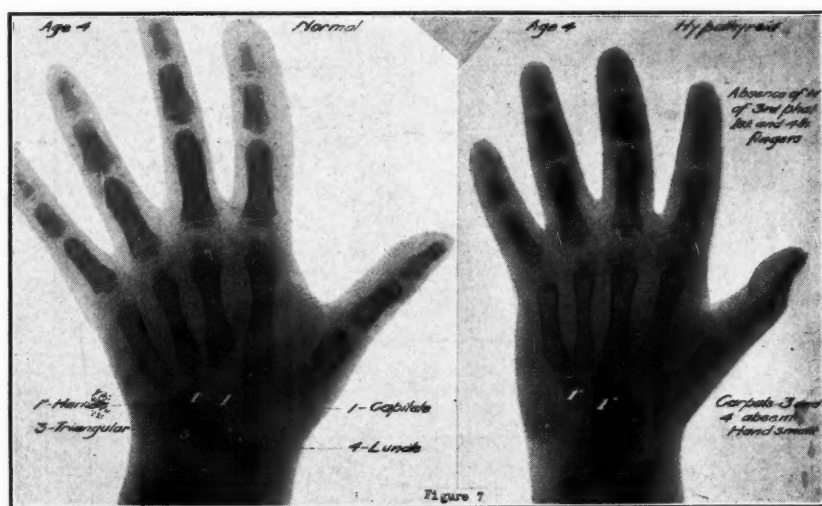


Fig. 7.

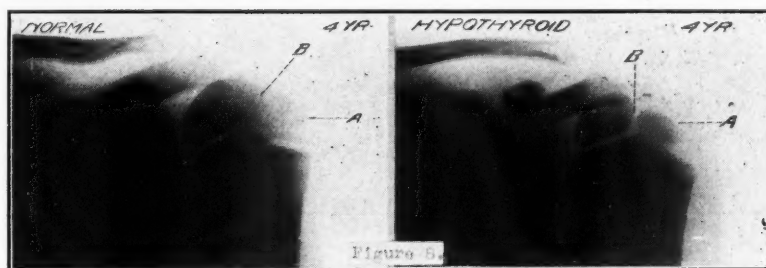


Fig. 8.—Aged 4. Normal shows (A) greater tubercle of humerus well developed; (B) partially fused with head of humerus. Hypothyroid shows (A) greater tubercle of humerus undeveloped; (B) absence of fusion of head and greater tubercle.

Aged five. In Fig. 14 the hypothyroid hand (plate No. 1) is developed to the age of two, with three carpal bones absent, the os triangularis, os lunatum, and navicular; while plate No. 2 shows a normal variation in the carpal development for the age of five, with no obvious endocrine dysfunction to account for the slightly advanced development. The heads of the metacarpals of the hypothyroid hand are developed to the age of three. It is to be noted in Plate No. 2 that all the carpal centers are present except the pisiform. The os triangularis and os lunatum are not fully developed for the age. The head of the radius is well developed.

Plate No. 1 (Fig. 15) is the elbow of a normal five-year-old child. Plate No. 2 is an elbow of a hypothyroid child of the same age. In the normal subject on the left the centers for the medial epicondyle and head of the radius are present, the latter just appearing, while the capitellum is well developed. In the hypothyroid subject there is an absence of these centers, together with underdevelopment of the capitellum. The difference in size and density of the bones is to be noted.

Fig. 16 is a comparison of the foot of a hypothyroid child of five years with that of a normal child of the same age. In the hypothyroid foot on the right the navicular and the heads of the second to the fifth metatarsal, and

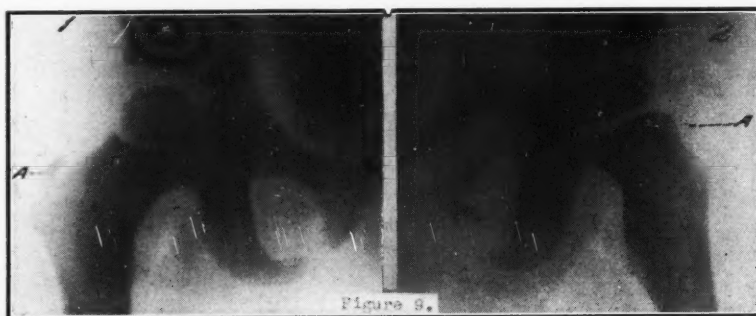


Fig. 9.—(1) Normal, aged 4. (A) Presence of greater trochanter of femur. (2) Hypothyroid, aged 4. (A) Absence of greater trochanter of femur.

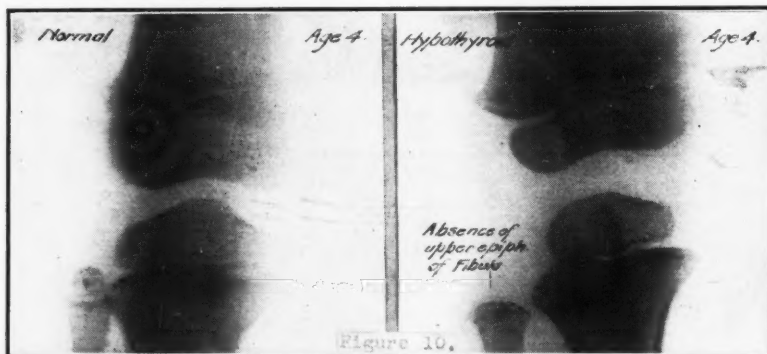


Fig. 10.

many of the phalangeal heads are absent. The second cuneiform is undeveloped. This foot represents about a three-year development.

Aged six. In Fig. 17 the same underdevelopment of the carpals is seen in the hypothyroid subject on the right as has been found at other ages. The navicular and the greater and lesser multangulars are absent, together with delay in development of the heads of the metacarpals and phalanges. The carpals present are developed to the age of four.

A case of suspected hyperpinealism at the age of six is contrasted with the normal condition for the same age in Fig. 18. The carpal bones, metacarpals, and phalanges are large, developed to about the age of fourteen, with beginning closure of all epiphyseal lines, particularly evident in the metacarpals. The head of the ulna, which is absent in the normal, is well

developed in the subject with suspected pinealism. This overdevelopment of the osseous system for the age is characteristic of this type of case. Early closure of the epiphyseal lines is a constant feature. The same advanced development may be seen in the other epiphyses about the body. It is unusual in these cases to find the epiphyseal lines open after the fifteenth year. The epiphyseal lines of the long bones normally close at about the eighteenth year.

In Fig. 19 are two elbows. No. 1 on the left is that of a normal subject aged six, while No. 2 on the right is that of a hypothyroid. In the normal elbow on the left the medial apicondyle is faintly visible, while the head of

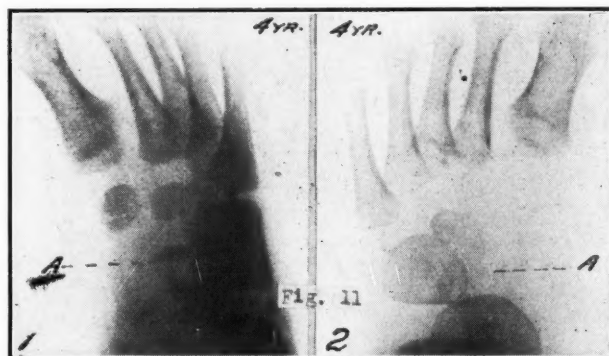


Fig. 11.—(1) Normal, aged 4. (A) Presence of navicular. (2) Hypothyroid, aged 4. (A) Absence of navicular. Absence of second cuneiform, with under-development of all tarsals.

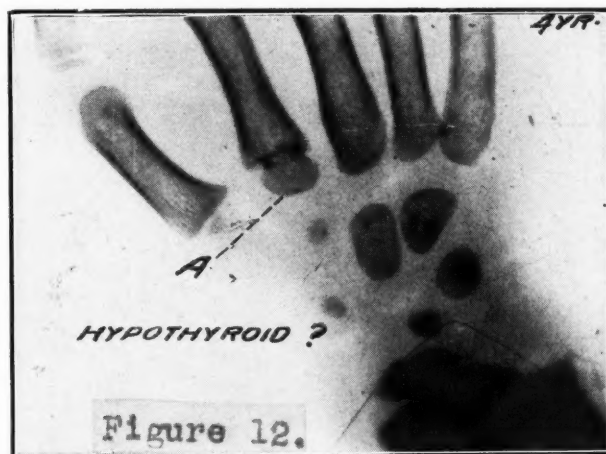


Fig. 12.—Aged 4. Clinical diagnosis, hyperthyroid, doubtful because of advanced carpal development for age. Presence of navicular and lesser multangular. (A) Accessory head of second metacarpal.

the radius is fairly well developed. These are absent in the hypothyroid elbow on the right. There is a general underdevelopment of the bones in the latter.

Aged seven. In Fig. 20 the hypothyroid subject on the right again demonstrates the underdevelopment of the carpals. The normal subject on the left shows all carpal centers present and well developed, with the head of the ulna likewise present. This is the earliest age at which the writers have found this center appearing, although most authorities state that it appears one or two years earlier. The hypothyroid subject on the right shows underdevelop-

ment of the carpal bones, which the writers have found so constantly in these cases. There is an absence of the head of the ulna. The hand is developed to about three years.

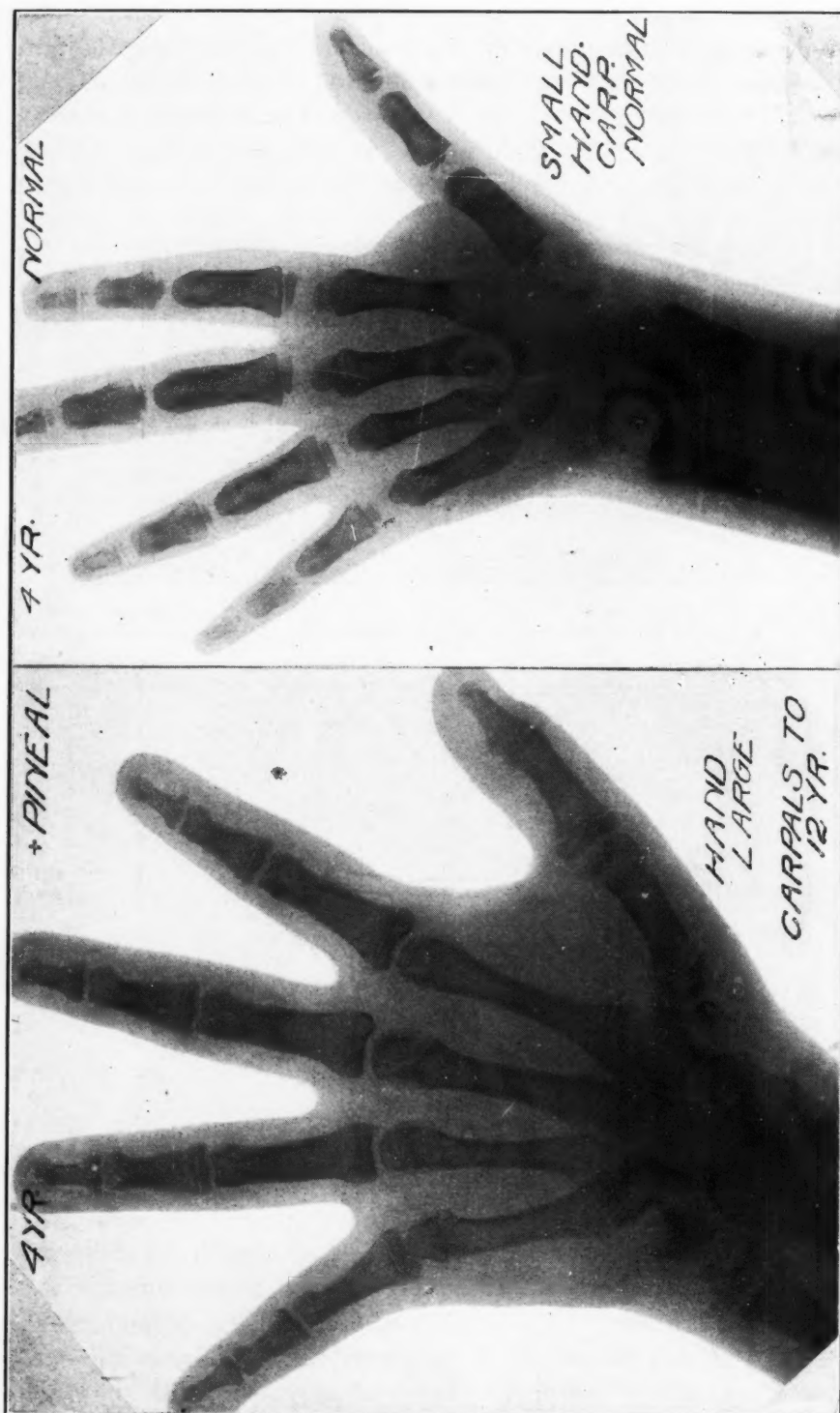


Fig. 13.

The carpal development in the hypothyroid hand in Fig. 21 at age seven might well be contrasted with that of the hypothyroid in Fig. 20. The differ-

ence between the two is evident. In that of Fig. 21 we have all the centers present for the age, but not developed to normal size. The head of the ulna is absent. The heads of the metacarpals and phalanges show the same underdevelopment. Clinically this case is one of hypothyroidism. The importance of radiographic examination in these cases of minor deficiency cannot be overemphasized, for where clinical characteristics are absent, it is frequently possible to make a diagnosis from the radiographic examination.

A radiogram of the pelvis of a hypothyroid subject, aged seven, contrasted with the normal pelvis in Fig. 22, shows separation of the pubis and ischium in the hypothyroid on the right, while fusion of the two bones is



Fig. 14.—(1) Hypothyroid, aged 5. Carpals for age absent—os triangularis, lunate, navicular, and greater multangular. Head of first metacarpal absent. (2) Advanced carpal development for age, lesser multangular present. Head of first metacarpal present.

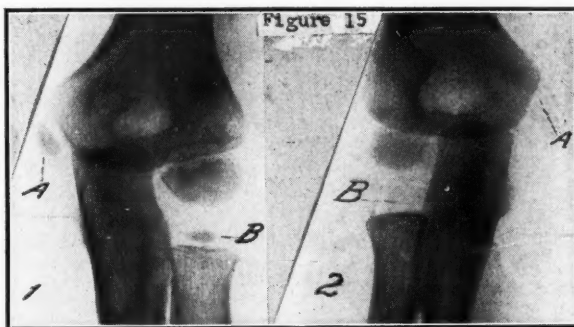


Fig. 15.—(1) Normal, aged 5. (2) Hypothyroid, aged 5. Note absence of internal condyle of humerus and head of radius in Fig. 2.

complete in the normal on the left. This fusion has been found to occur normally at this age. The hypothyroid pelvis represents a development of about five years.

Aged seven to nine. The radiogram in Fig. 23 shows the variations that occur in the appearance of the center for the epiphysis of the calcaneus. This normally appears between seven and nine years. In A, at the age of seven, it is absent, while in B, at the age of nine, the center is just appearing, being well developed in C, at the age of seven, probably appearing at about the sixth year in this patient. In this last case the other epiphyseal centers about the body were developed to a degree normal for the age.

Aged nine and ten. The pisiform is present in No. 1 (Fig. 24), the hand

of a patient aged nine with thyro-pituitary insufficiency, while it is absent in the hypothyroid subject (No. 2) aged ten. This, together with a general tendency toward slight advancement in the carpal development, has been found frequently in these cases. The carpal bones in thyro-pituitary insuffi-

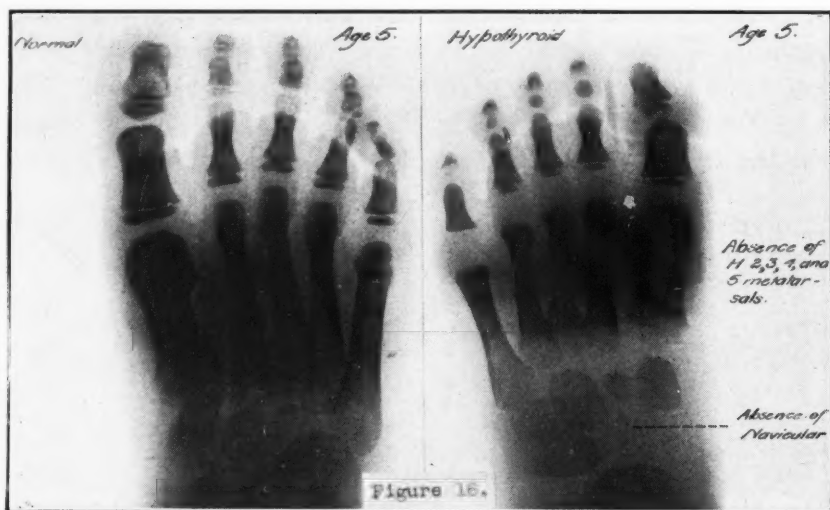


Fig. 16.

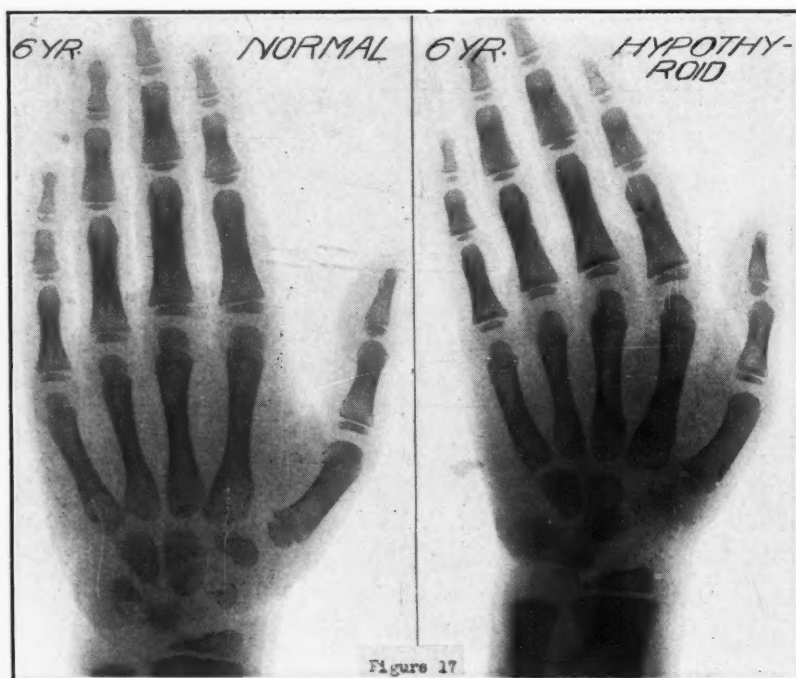


Fig. 17.

ciency are denser than those of the hypothyroid patient, with more tendency to massing. The pisiform appears normally between nine and eleven years.

The elbow of the thyro-pituitary subject (Fig. 25, No. 1) at nine years gives evidence of the tendency to slightly advanced osseous development, as

has been seen in Fig. 24. This is apparent in the appearance of the lateral epicondyle of the humerus and well developed olecranon and trochlea, while in the normal child (No. 2) of ten years the center for the lateral epicondyle is just appearing. This has been found present normally from ten to twelve years. The center for the olecranon is about the size for the age, the center for the trochlea being absent. This last appears about the tenth year.

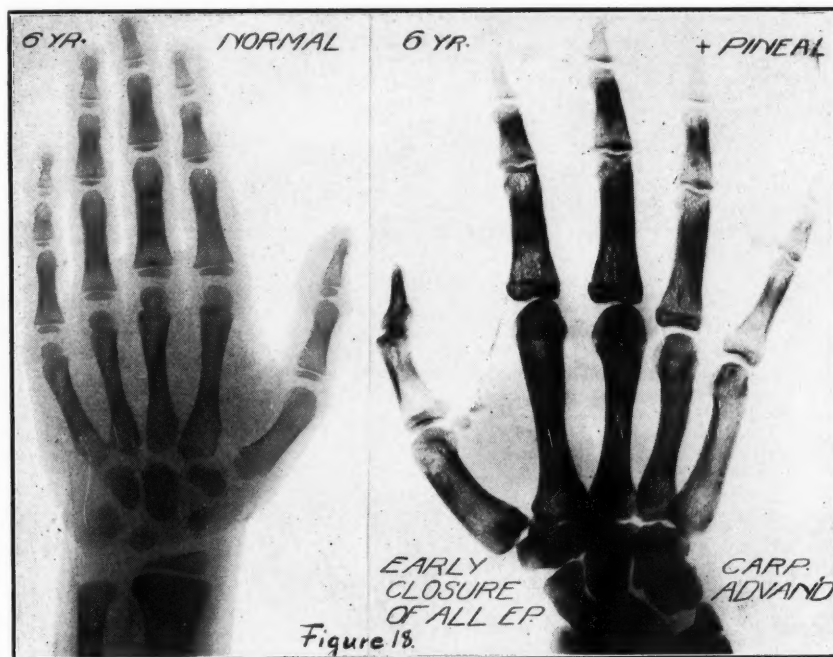


Fig. 18.

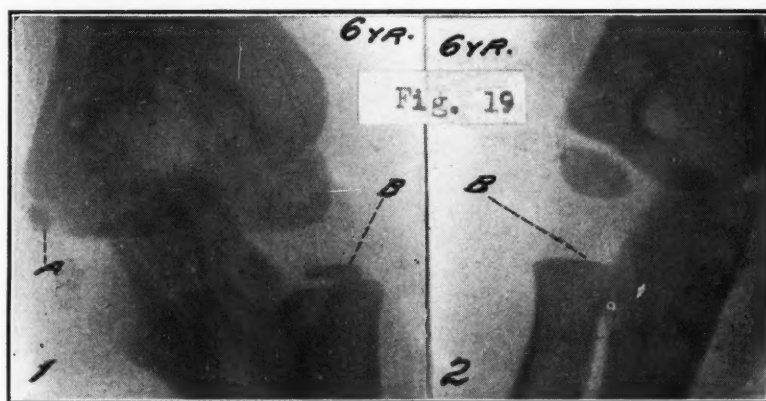


Fig. 19.—(1) Normal, aged 6. (A) Appearance of medial epicondyle. (B) Presence of head of radius. (2) Hypothyroid, aged 6. (B) Absence of head of radius. Absence of medial epicondyle. Note difference in size of bones.

Aged ten and eleven. In Fig. 26 the elbow of a subject, aged eleven, with a pluriglandular (pituitary-thyroid) deficiency is compared with the elbow of a normal child, aged ten. We have mentioned above in Figs. 24 and 25 that the thyro-pituitary subject shows a tendency to a slight advance in the osseous development, as contrasted with the pure thyroid. This advanced devel-

opment has been seen chiefly in the carpals and centers for the long bones. The subject with pituitary-thyroid deficiency,* on the other hand, shows a tendency to a slight delay in the development of these centers. These points have been brought out in a limited series of cases, and suggest further study,

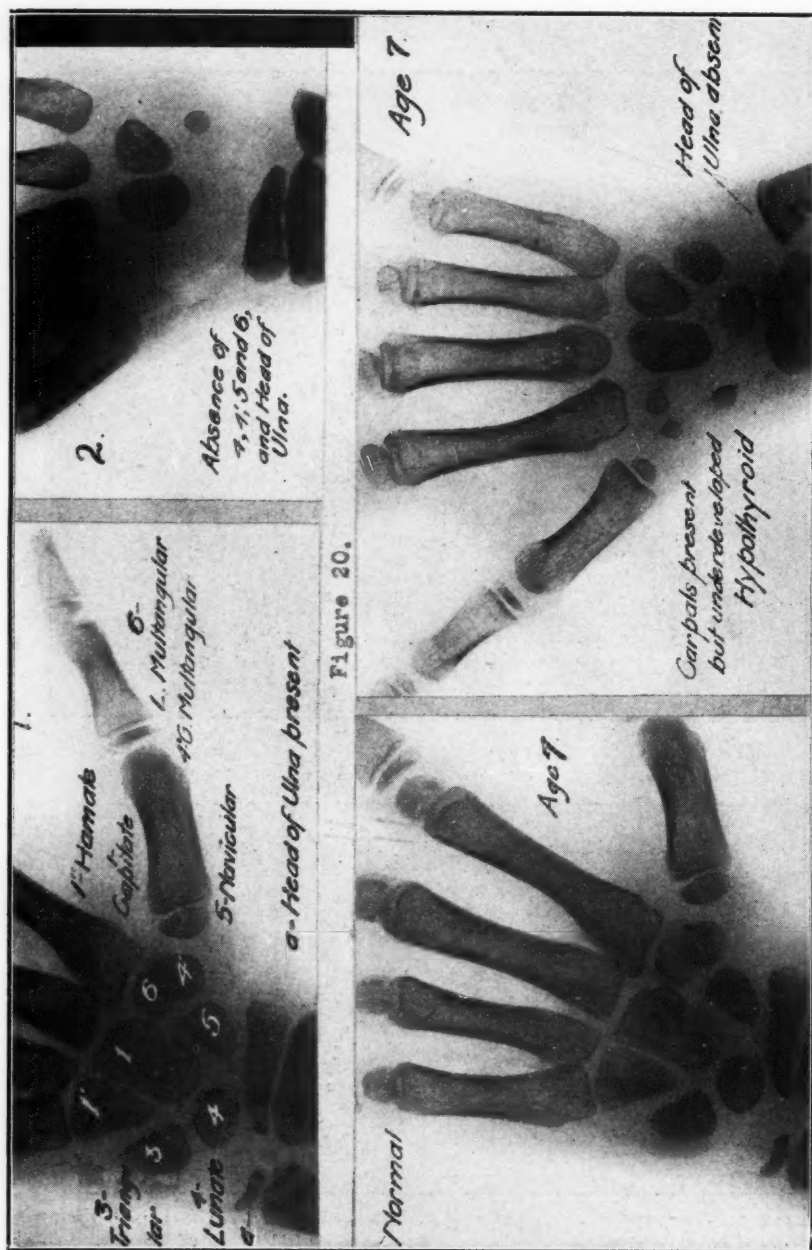


Fig. 21.

Figure 20.—(1) Normal, aged 7. All carpals present and well developed. Hypothyroid, aged 7. Three carpals present; carpals absent Nos. 4, 4', 5 and 6. Head of ulna absent.

Fig. 21.—Comparison of hypothyroid, age 7, with normal, age 7, on left.

with the confirmation of additional cases. The delayed development is seen in Fig. 26, in which the trochlea is absent in the pituitary-thyroid, and well developed in the normal subject. The olecranon is undeveloped in the pluri-

*The distinction between thyro-pituitarism and pituitary-thyroidism lies in the fact that in the former there is early hypothyroidism, with a subsequent pituitary deficiency, while in the latter the pituitary deficiency antedates and is complicated by the thyroid deficiency.

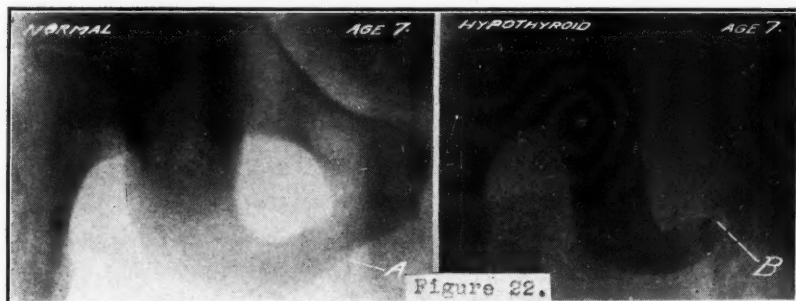


Fig. 22.—Normal shows (A) Union of pubis and ischium. Hypothyroid shows (B) non-union of pubis and ischium.

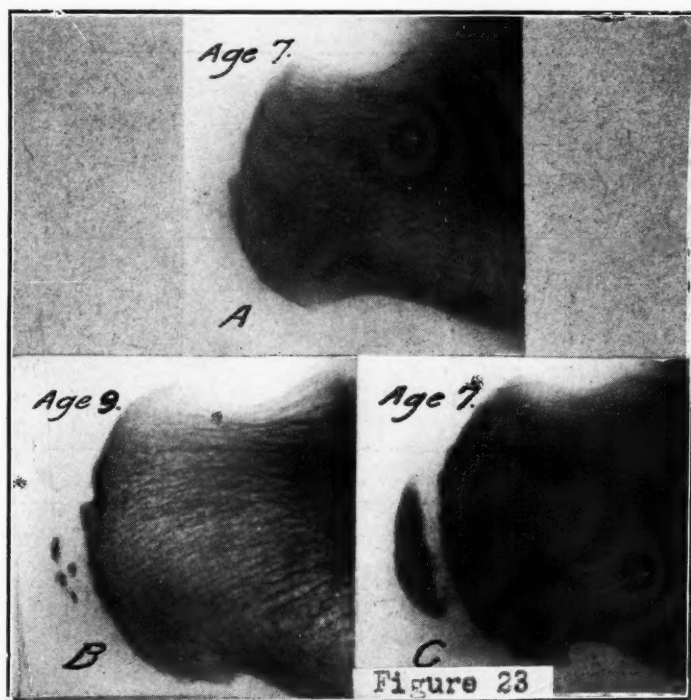


Fig. 23.—(A) Normal, aged 7. Absence of epiphyseal head of calcaneus (os calcis). (B) Aged 9. Appearance of epiphyseal head of os calcis (occurs normally at 7 to 8). (C) Aged 7. Presence of head of os calcis.

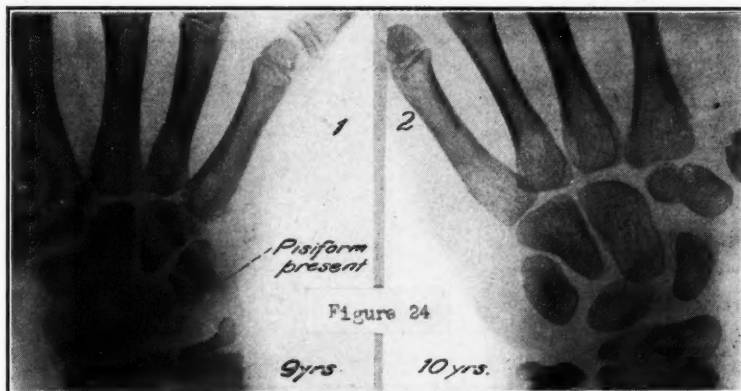


Fig. 24.—(1) Thyro-pituitary insufficiency (aged 9). Note presence of pisiform. (2) Hypothyroidism (aged 10). Note absence of pisiform.

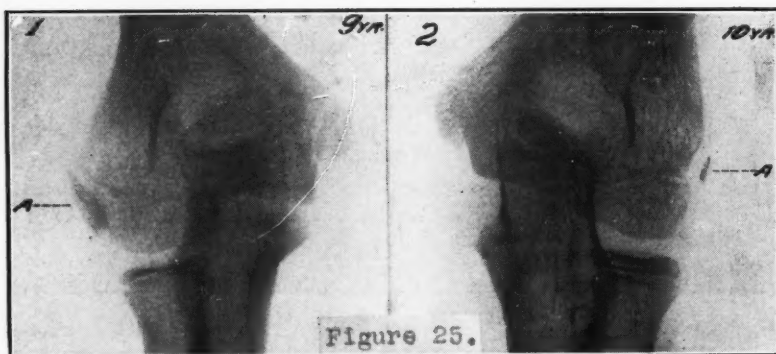


Fig. 25.—(1) Thyro-pituitary, 9 years. (A) Well developed lateral epicondyle. Note well developed olecranon. (2) Normal, 10 years. (A) Appearance of lateral epicondyle. Olecranon small.

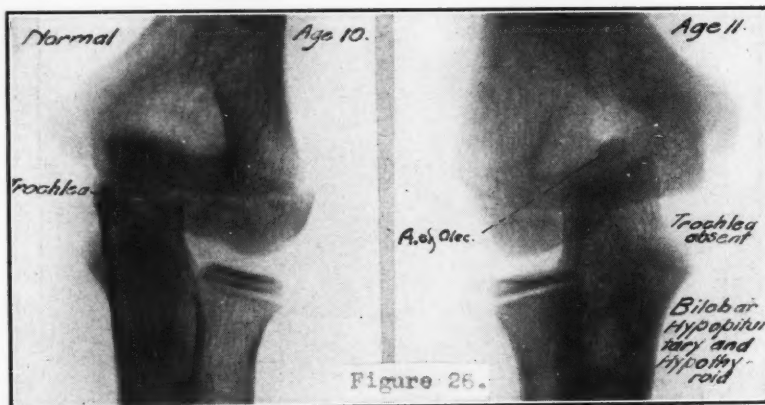


Fig. 26.

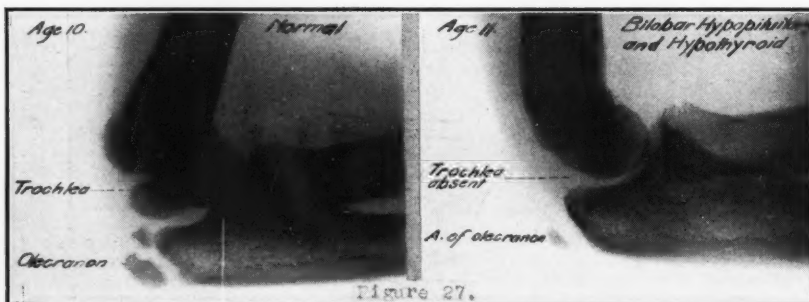


Fig. 27.

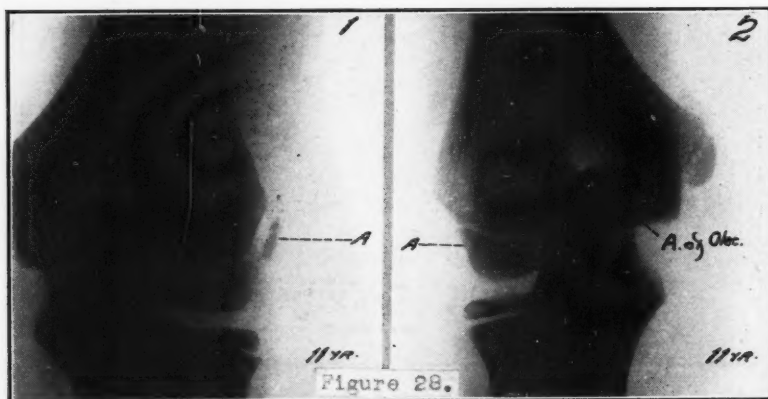


Fig. 28.—Aged 11. (1) Normal. (A) Appearance of lateral epicondyle of humerus. (2) Pluriglandular, bilobar hypopituitarism, with hypothyroidism. (A) Absence of lateral epicondyle of humerus.

glandular, while absent in the normal subject, appearing at about the tenth year.

Fig. 27 shows a lateral view of the elbows of the same subjects as seen in Fig. 26, in which the trochlea and olecranon are well developed in the normal elbow on the left, while the trochlea is absent in the thyro-pituitary elbow on the right, and the center for the olecranon has just appeared.

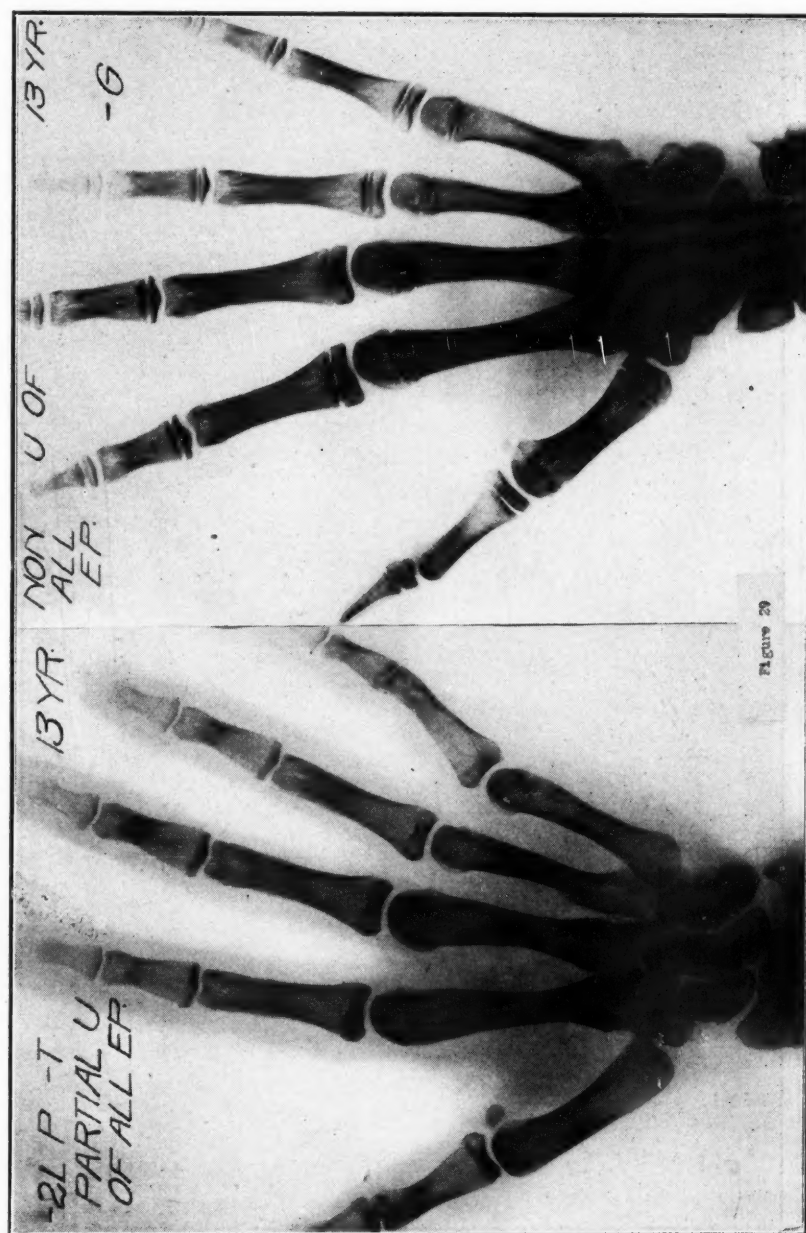


Fig. 29.

Aged eleven. In the normal elbow (No. 1, Fig. 28) the lateral epicondyle of the humerus has just appeared, while the same center is absent in the pituitary-thyroid elbow on the right. The olecranon is well developed in the normal and small in the pluriglandular elbow. The trochlea is also well developed in the normal and absent in the pluriglandular elbow.

Aged thirteen. In the above Fig. 29 the pluriglandular (pituitary-thyroid) hand shows beginning closure of the epiphyseal lines of the metacarpals and phalanges. The heads of the metacarpals normally fuse between fourteen and fifteen years, while the heads of the phalanges fuse at fifteen years. The



FIGURE 30.

Fig. 30.

hypogonad hand on the right has all epiphyseal lines clearly open, being much larger than the pituitary-thyroid hand, with more pronounced development of the metacarpals and phalanges. The striking difference, however, lies in the open epiphyseal lines of the hypogonad hand, a fact which is brought out constantly in this type of case in later years.

Aged thirteen and fourteen. The same pituitary-thyroid hand as seen in Fig. 29 is compared in Fig. 30 with a hypothyroid hand at fourteen years. The pituitary-thyroid hand on the left is much larger than the hypothyroid hand on the right, showing beginning closure of the epiphyseal lines of the metacarpals and phalanges. The carpals are well developed for the age. The appearance of the carpal centers after nine to eleven years, at which time the pisiform appears, is not of particular value in the diagnosis of endocrine dysfunction. However, the underdevelopment of the carpal centers, as is seen in the hypothyroid hand at fourteen years, together with a delayed closure of the epiphyseal lines, can be used as a diagnostic sign.

Aged fourteen. The radiogram shown in Fig. 31 is that of a shoulder, with the center for the acromion plainly evident. This is said to arise from

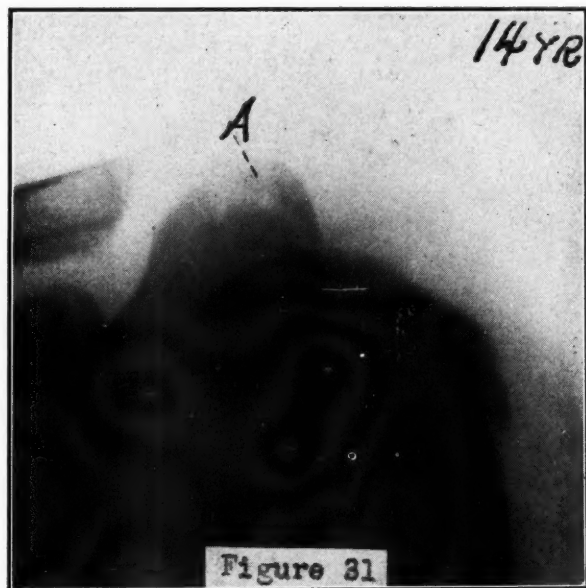


Fig. 31.—Normal, aged 14. (A) Appearance of acromion.

two centers. The writers have not been able to confirm this. The normal appearance for this center is from the fourteenth to the fifteenth year.

Aged fifteen and sixteen. On the right, the hand of the subject with suspected hyperpinealism at the age of fifteen in Fig. 32, presents a development of all bones of the hand far in advance of the normal hand, while the hypothyroid hand of a patient aged sixteen on the left confirms the earlier signs of general osseous underdevelopment in this type of case. The hands resemble each other very closely in size, but differ in the density of the bones and most particularly in the fact that the former at fifteen years has all epiphyseal lines closed, while the latter at sixteen has epiphyseal lines open. There is complete union of the heads of the radius and ulna in the former. The head of the radius does not normally close until the seventeenth year, and that of the ulna, between the eighteenth and twentieth years. This early closure extends to all the long bones. The massing of the carpals is more pronounced in the subject with suspected hyperpinealism than in the hypothyroid subject.

In Fig. 33 the same suspected hyperpineal hand at the age of fifteen is contrasted with the hand of a subject with anterior lobe pituitary deficiency at the age of twenty-one. The characteristics of the former have been described under Fig. 32. The hand of the patient with anterior pituitary deficiency is

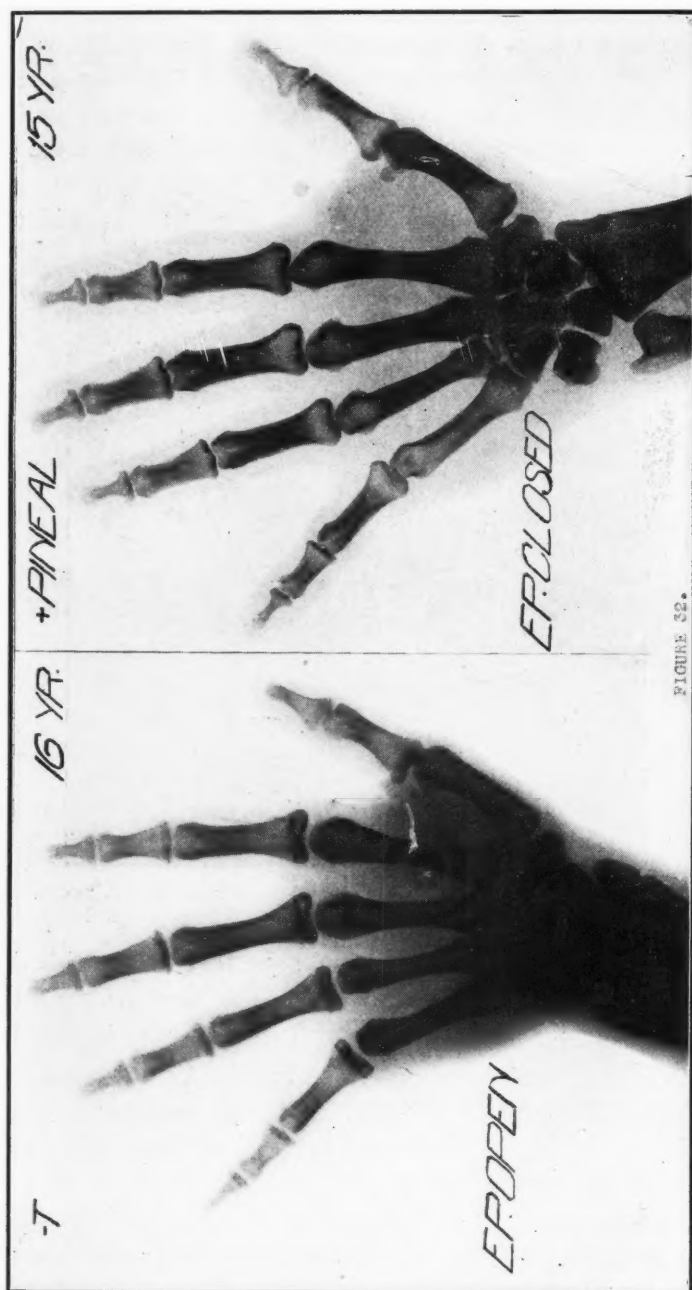


FIGURE 32.

Fig. 32.

small, but slightly larger than the pineal hand. There is, however, a persistence of all epiphyseal lines in the former, including the epiphyses of the radius and ulna, which normally close between seventeen and nineteen years. We have a contrast of early closure of epiphyseal lines and delayed closure in two subjects who from the standpoint of stature resemble each other closely

but who differ widely in many important characteristics, such as mentality, the development of the primary and secondary sexual characters, etc. The etiologic factor of the decreased stature in one is essentially different from that in the other. The early closure of epiphyseal lines in the suspected



Fig. 33.

hyperpineal hand prevents normal growth, while the absence of the anterior lobe pituitary secretion does not permit the normal growth of the long bones in this type of case, so that the stature remains small in spite of the fact that all epiphyseal lines remain open.

In Fig. 34 the shoulder of a normal subject aged fifteen is contrasted with

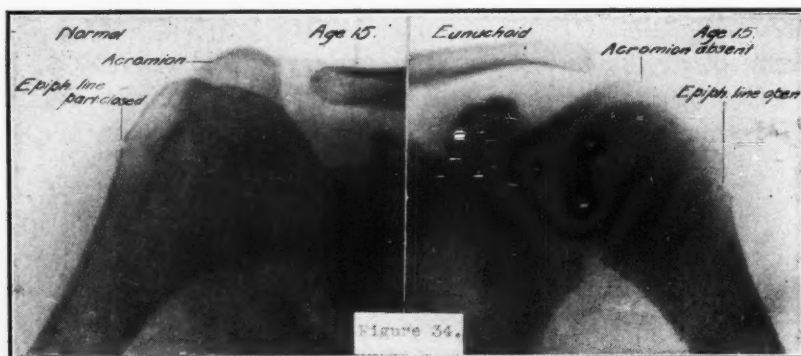


Fig. 34.

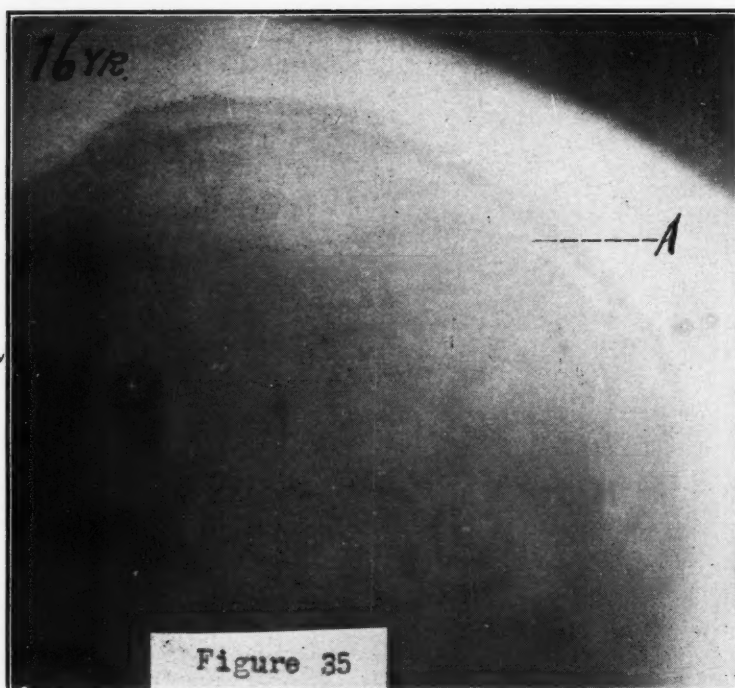


Fig. 35.—Bilobar hypopituitarism, aged 16. (A) Crest of ilium well developed (normal at 15).

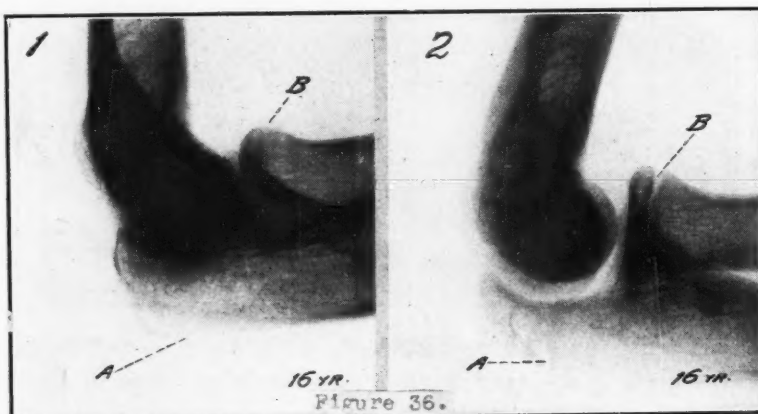


Fig. 36.—Aged 16. (1) Normal, showing (A) olecranon closed; (B) head of radius closed. (2) Hypopituitarism, preadolescent, showing (A) olecranon open; (B) head of radius open.

the eunuchoid shoulder of the same age. The center for the acromion is present in the shoulder on the left, and there is beginning closure of the epiphyseal line of the head of the humerus. Normally the head of the humerus is completely fused with the shaft at the eighteenth year. The eunuchoid subject



Fig. 37.

on the right shows an absence of the epiphysis of the acromion, with the epiphyseal line of the head of the humerus completely open. This delay in the closure of the epiphyses is, as mentioned above, characteristic of the hypogonad or eunuchoid type.

Aged sixteen. The center for the crest of the ilium is shown well devel-

oped in Fig. 35. This normally appears at the fifteenth year, together with the other secondary centers of the os coxae. Other osseous findings at the age of fifteen to sixteen are the appearance of the sternal end of the clavicle, union of the heads of the phalanges of the hand, and union of the primary centers of the os coxae.

In Fig. 36 is seen other evidence of the delay in the closure of the epiphyseal lines in the subject with anterior lobe pituitary deficiency. The normal elbow on the left of a subject aged sixteen shows complete closure of the olecranon and practically complete closure of the epiphyseal line of the head of the radius. The elbow of the patient with anterior lobe deficiency shows

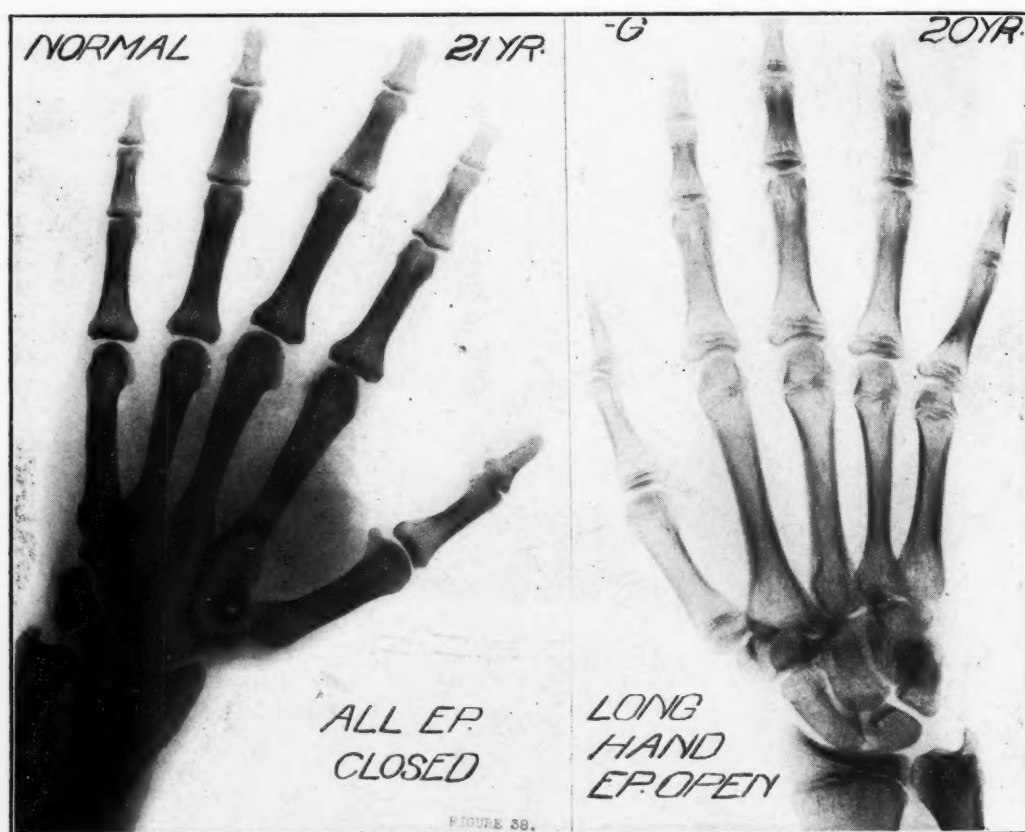


Fig. 38.

the epiphyseal lines open. The same delay in closure is seen in the distal epiphysis of the humerus, this normally closing at about sixteen years.

Aged eighteen. The subject with pluriglandular deficiency (pituitary-thyroid) in Fig. 37 shows a delay in the closure of all epiphyseal lines, the epiphyseal lines of the metacarpals and phalanges normally closing at fifteen to sixteen years, while the epiphyseal lines of the radius and ulna, which are still present in this hand, normally close between the seventeenth and nineteenth years. The hypothyroid hand on the left shows closure of all epiphyseal lines, which is normal for the age. The hands resemble each other very closely in size.

Aged twenty. The hypogonad hand on the right in Fig. 38 shows a gen-

eral increase in length, with a persistence of the epiphyseal lines of the metacarpals, phalanges, radius, and ulna. The hand is slender, the increase in length being accounted for by the failure of the epiphyseal lines to close, as

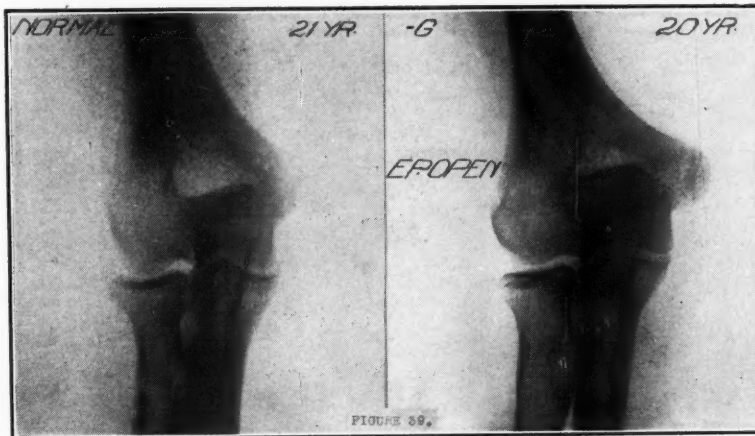


Fig. 39.



Fig. 40.

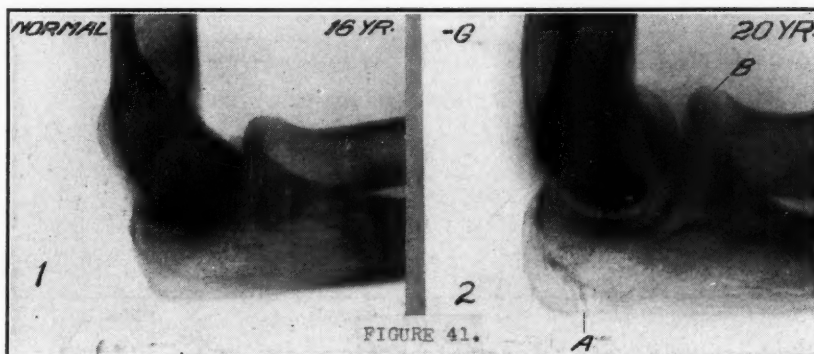


Fig. 41.

they do normally between the fifteenth and nineteenth years. The normal hand on the left shows all epiphyses closed.

The radiograms of the elbows of the same subjects as seen in Fig. 38 are contrasted in Fig. 39. All epiphyses are closed in the normal elbow. There is complete fusion of the separate centers of the distal extremity of the hu-

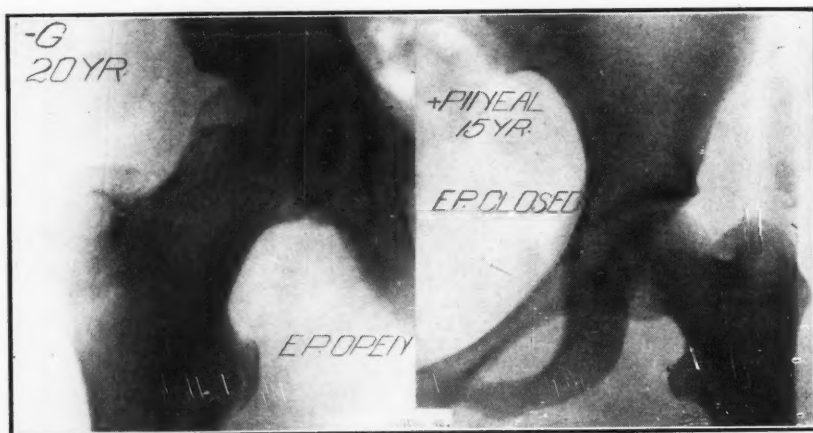


Fig. 42.

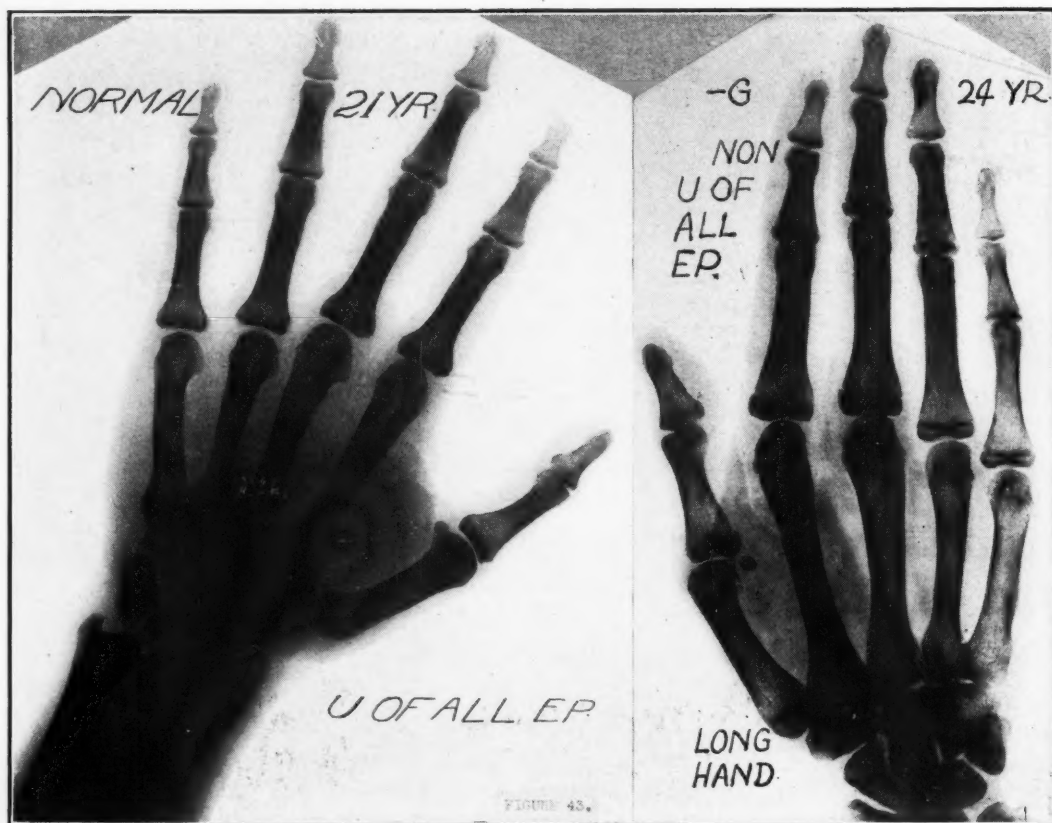


Fig. 43.

merus, together with fusion of the distal extremity of the humerus with the shaft. The olecranon and the head of the radius are closed, these normally closing from the sixteenth to the nineteenth years. These epiphyses are frankly open in the hypogonad elbow on the right.

The radiograms in Fig. 40 are those of the same hypogonad subject taken after an interval of one year. The outstanding feature is a persistence of all epiphyseal lines of femur, tibia, and fibula at the age of twenty. There apparently has been no tendency for the epiphyseal lines to close in the interval elapsing between the first and second radiograms, taken at nineteen and twenty years respectively. The delay in closure of the epiphyseal lines is seen in all the long bones, with resultant increase in stature due to a preponderance of long bone development.

In Fig. 41 the epiphyses of the elbow of a hypogonad subject aged twenty

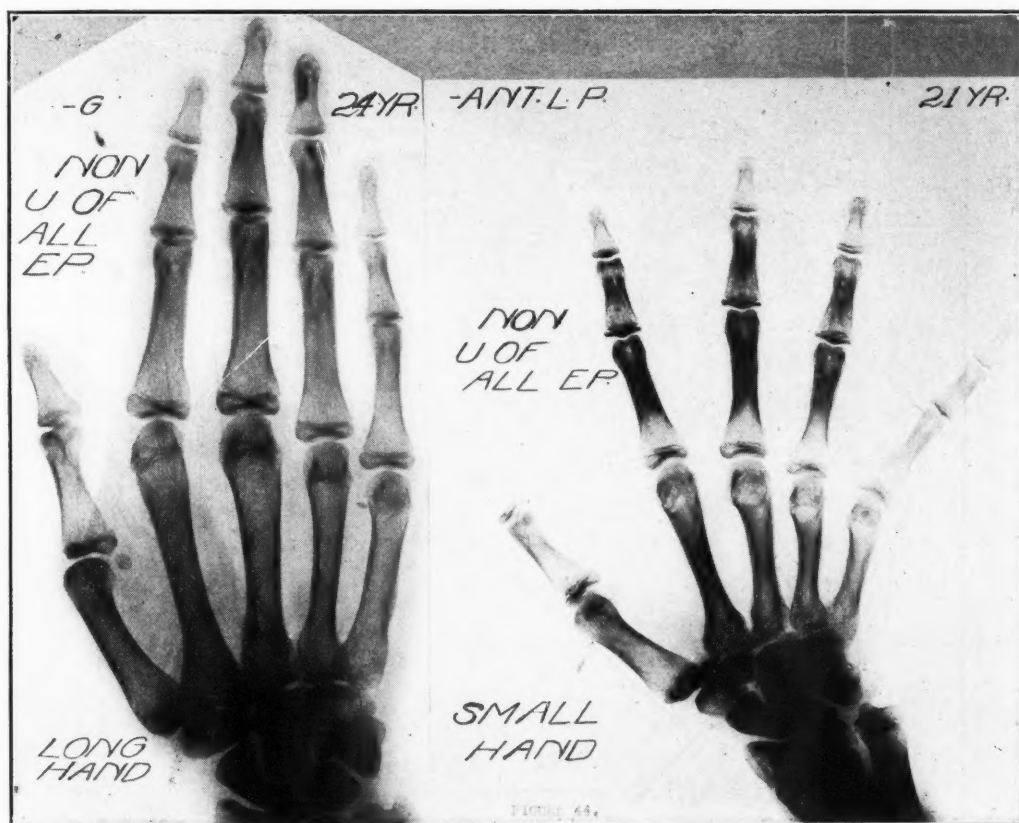


Fig. 44.

are contrasted with those of a normal subject aged sixteen. In the latter the epiphysis of the olecranon is closed and the head of the radius practically closed. In the former the epiphyses are open.

The pelvis in Fig. 42, showing the head of the femur in a hypogonad subject of twenty, gives additional evidence of the delayed closure of the epiphyses, and contrasts strongly with the suspected hyperpineal subject of fifteen on the right, in which all epiphyses are closed, a point emphasized in the earlier figures. The clinical types differ markedly in somatic and genital development.

Aged twenty-four. In Fig. 43 the hand of a hypogonad subject aged twenty-four is contrasted with that of a normal subject aged twenty-one. The same characteristics of hypogonad development, the size and type of

hand, the open epiphyses, etc., as manifested in the above comparisons, are evident in the picture on the right. It is to be noted that the hypogonad patient in this figure is four years older than the hypogonad patient shown previously, and yet there is no evidence of closure of the epiphyses.

In Fig. 44 we compare a hypogonad hand, aged twenty-four, with that in a case of anterior lobe pituitary deficiency at the age of twenty-one. The difference in size of the hands is manifest. In the hypogonad subject we have the slender, artistic "eunuchoid" hand, while in the anterior lobe deficiency we have the small, fragile hand, the type "en petite." In both, we have

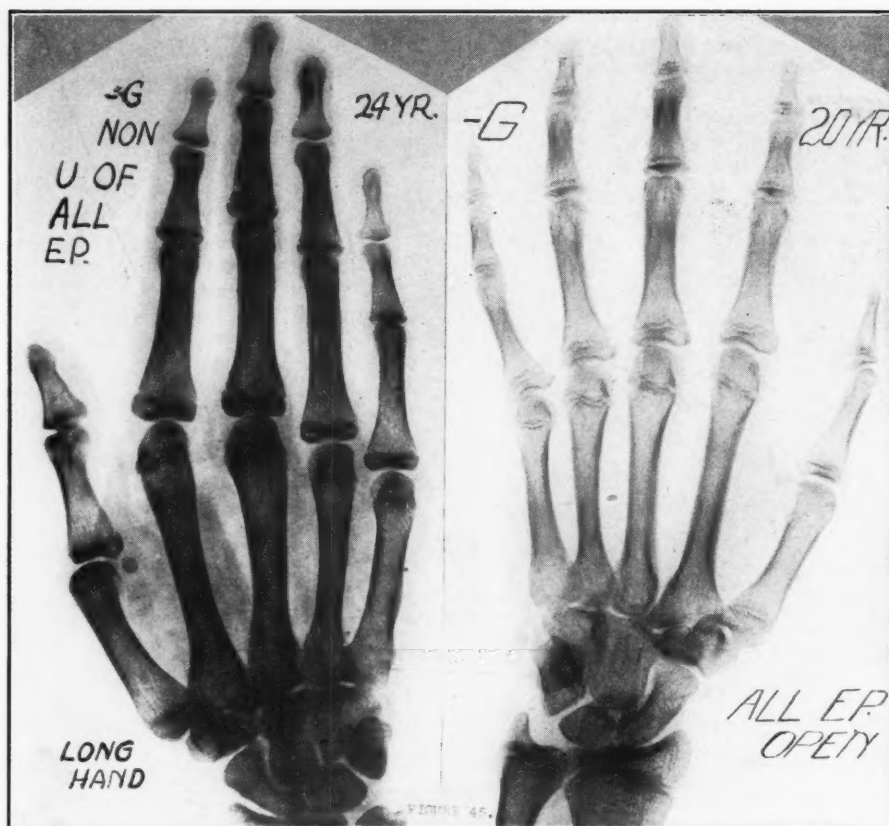


Fig. 45.

a persistence of the epiphyseal lines. In the former we have overgrowth of the long bones, and in the latter underdevelopment of the long bones. Clinically the types are distinct. The size of the hand, apart from the stature and general bodily configuration, is sufficient to differentiate the two types, although the epiphyseal lines are persistent in both.

Aged twenty-four and twenty. A comparison of two hypogonad hands is seen in Fig. 45, both hands of a classical type, with the epiphyseal lines clearly open three to six years beyond the normal age.

Aged twenty-one. The radiograms of the knee joints of a subject aged twenty-one with anterior lobe pituitary deficiency and a subject with hypogonadism aged twenty display the characteristics which have been evidenced

in the above figures, in the persistence of the epiphyseal lines seen in the hands and other long bones. There is a marked difference, however, in the size of the long bones and the size of the corresponding epiphyses.

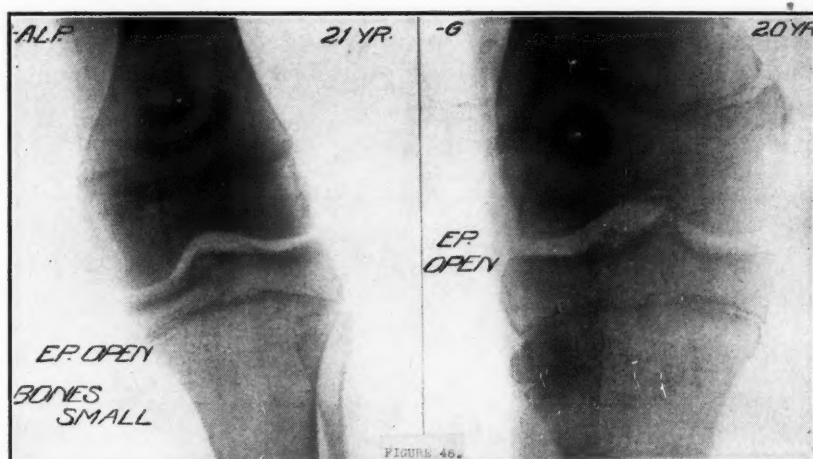


Fig. 46.

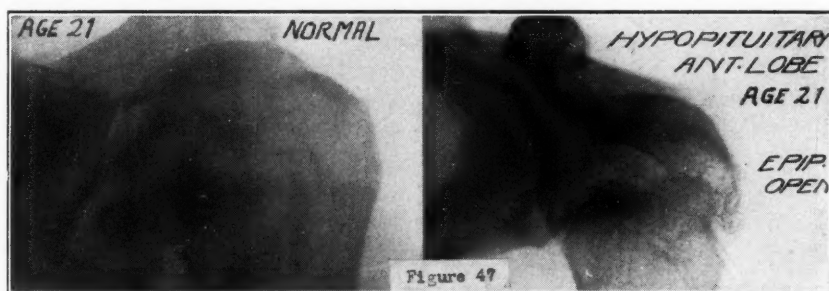


Fig. 47.—Normal, aged 21, showing epiphyseal line of humerus closed. Hypopituitary, anterior lobe deficiency, aged 21, showing epiphyseal line of humerus open.

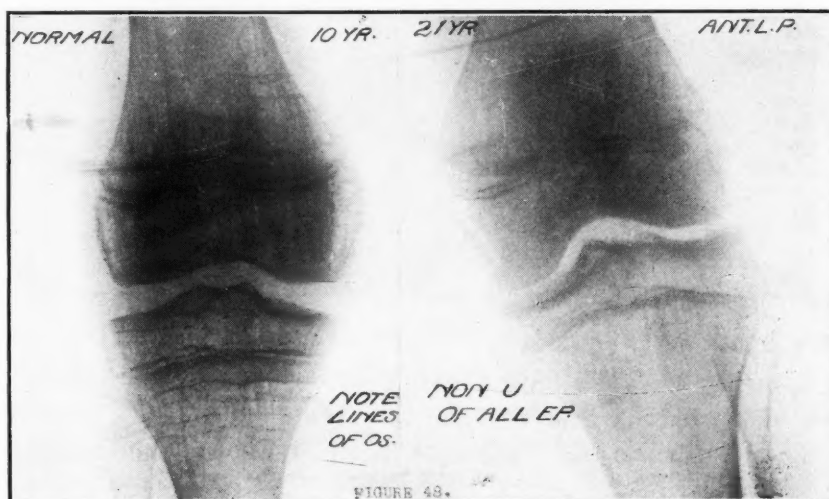


Fig. 48.

The normal subject aged twenty-one in Fig. 47 shows complete closure of the epiphyseal line of the head of the humerus, this normally closing about

the eighteenth year. In the anterior lobe pituitary deficiency there is a persistence of the epiphyseal line of the head of the humerus.

In Fig. 48 the osseous development of the knee in anterior lobe deficiency at the age of twenty-one is seen to be that of about ten years. There is a close resemblance between these radiograms, as evidenced in the epiphyseal lines, size of the bones, etc. The lines of ossification are to be noted in the normal ten-year knee on the left.

Acromegaly. In Fig. 49 note the characteristic spade hand of the acro-



Fig. 49.

megalic, showing the well known tufting of the distal phalanges, with hook formation of the distal phalanx of the thumb, the prominence of the tuberosities of the metacarpals and phalanges, the increase in breadth of all bones, and the thickening of the cortex.

The radiogram (Fig. 50) of the sella turcica of the same patient whose hand is presented in Fig. 49 shows that the sella is definitely enlarged, without evidence of erosion. There is a thickening of the bones of the skull, with an enlargement of all sinuses, seen particularly in the frontal sinuses.

Aged twenty-five. The hand of a giant in Fig. 51 shows marked increase in growth of all bones. The hand is extremely large, the palm being narrow in proportion to the length of the fingers. It is to be noted that there is absence of tufting of the distal phalanges, differentiating it from the hand of the acromegalic in Fig. 49. The proportions and size also serve as differentiating factors. All epiphyseal lines are closed, although in some of the heads of the phalanges a faint epiphyseal line may be seen, suggesting delayed closure, with production of increase in length of the long bones.

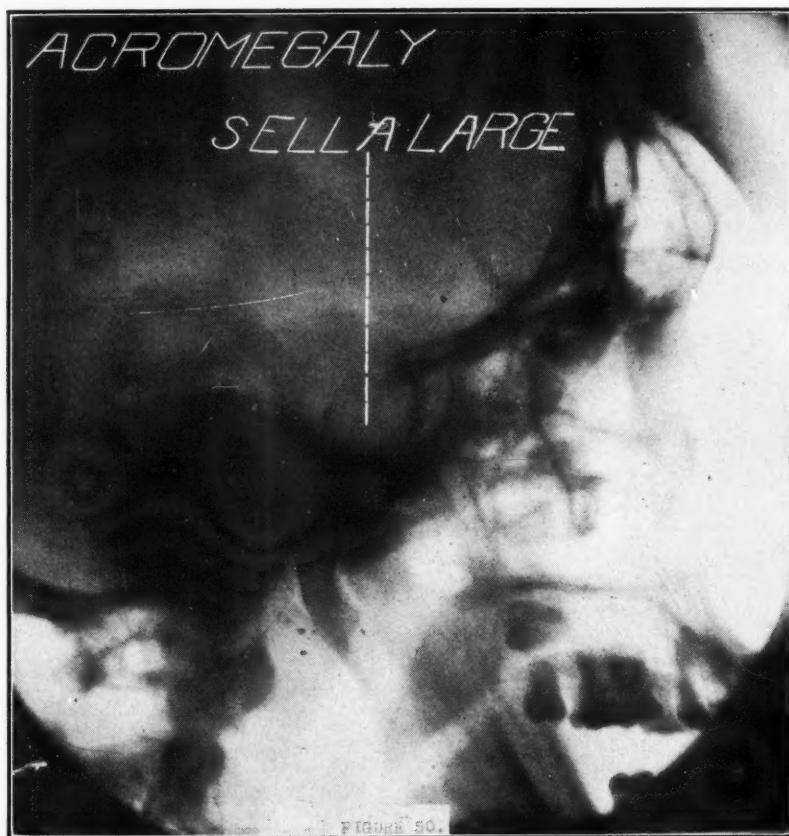


Fig. 50.

CONCLUSIONS

(1) The general diagnostic information derived from the roentgenologic comparison of endocrinopathic and normal subjects has led the writers to believe that the radiologic signs offer encouraging prospects of being of more value than the basal metabolism, blood chemistry, and other so-called specific and laboratory determinations.

(2) Retardation of development of all the bones of the osseous system, not only of the carpals, in uncomplicated hypothyroidism can be demonstrated roentgenologically in all ages up to that of completion of normal skeletal growth. This will be an additional aid to diagnosis in those cases already beyond the age of normal carpal development upon which basis

hitherto has depended the roentgen picture of osseous change indicative of hypothyroidism.

(3) Hypogonadism and eunuchoidism have consistently shown a definite late fusion of the epiphyseal ends of the long bones. While this has been suspected clinically, we are unacquainted with any definite roentgenologic



Fig. 51.

demonstration of these abnormalities in secondary hypogonadism. The late closure of the epiphyseal ends in the presence of an active hormone from the anterior lobe of the hypophysis explains the *overgrowth of the long bones* in these subjects.

(4) In anterior lobe pituitary insufficiency in which there is a primary deficiency of the anterior lobe and a secondary deficiency of the generative

organs, there has been found uniformly present a late closure of the epiphyseal ends of the long bones, *associated with undergrowth* of these bones. The reason for the undergrowth of the long bones in the presence of the open epiphyseal ends in this disorder is the *absence of the hormone from the anterior lobe of the hypophysis*.

(5) In the pluriglandular syndrome, the development of the osseous system as demonstrated roentgenologically is very difficult to interpret. From the studies of our cases thus far, we are of the impression that the following facts obtain: (a) In the thyro-pituitary disorder there is an advance of the carpal and long bone nuclei development over that of pure hypothyroidism unassociated with pituitary disorder. (b) In pituitary-thyroidism, there is a retardation of the appearance of the osseous nuclei, as well as of the fusion of the epiphyseal ends of the long bones, more marked than that in pure hypothyroidism or in the normal. (c) The markedly heterogeneous pictures presented in the multiglandular syndromes will depend upon the sequence in which the various disorders were superimposed upon each other. For this reason, the combination of the same glandular disorders might present entirely different radiographic pictures of the osseous development at the same age, depending upon the order in which the various glands might have become involved.

(6) In the less frequent but very instructive condition of *pubertas praecox* (suspected pinealism), the most unusual advancement in development of the bone nuclei and early fusion of the epiphyseal lines was found. The four cases studied confirmed our earlier belief relative to the effect of gonad hormone upon the osseous growth and development, and were a convincing confirmation of the exactly opposite picture consistently present in the hypogonad.

(7) Thymo-lymphatism in the few cases studied apparently presented much the same osseous retardation as mild hypothyroidism. The osseous development in positive cases of enlarged thymus should be more thoroughly studied, with the view of clearing up this much mooted point of the relation of thymus function to osseous development.

TECHNIC SUGGESTIONS RELATING TO APPLIANCES*

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THIS clinic was given, hoping it would be helpful to other orthodontists, as the suggestions listed below have been very helpful to the author. Briefly stated, these are:

1. Lingual lateral loop spring for expansion.
2. Hook for intermaxillary elastics which prevent rubbers from unhooking.
3. Use of paraffin to prevent irritation of buccal tubes, etc., when first inserted.
4. Uses of ligatures for apical movement of the four anterior teeth.
5. Balling end of wires, thus reducing irritation.
6. Solder used in wire form, and fluxed over its entirety.
7. Wires bent by heat (both lingual and labial arches).
8. Grooving end of tweezers, thus preventing side slipping of wires.
9. Wetting of ligatures just before tying knots.
10. Using two valves on air and gas connections to orthodontic burner and blowpipe.

The Lateral Loop Spring.—

Object: expansion in canine and premolar region.

Advantage: stable—adjusted easily—positive in action.

Construction: The lingual arch is made by any method desired, but one which is removable and adapted high in the palate of the maxilla, and conveniently low in the mandible is most desirable. The high palatal arch also prevents lipping. A small speck of solder is placed on the wire opposite the second premolar (either side). A 23-gauge spring wire of any reliable make is extended from the arch wire to the cervix of the second premolar, extending anteriorly along the gingiva approximately two millimeters beyond the first premolar, recurved (Fig. 1,b), and bent at right angles at the interproximal of the two premolars (c) to engage the arch wire at (d), and soldered. The shape and length of the spring is made before soldering, after which it is held in place and the distal end soldered, and the rest of the spring is adapted with heat and slate pencil, eventually soldering the opposite end. Short finger springs may be added when desired. The loop springs have the advantage over the plain finger springs used for expansion, in that they are not displaced by forces in mastication, nor by the tongue.

To adjust: Fig. 1. The wire is straightened at angle (c) by gripping with a pair of narrow flat-nosed pliers. This forces the wire (a-b) laterally and occlusally, which may necessitate bending the wire (a-b) gingivally. After

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the wire (b-c-d) has been straightened, additional expansion can be gained by rebending the angle (b), shortening wire (a-b) and lengthening wire (b-d), as this wire (a-b) was made overlength originally. Likewise angle (a) can be rebent to lengthen wire (a-e) to gain expansion in the second premolar region. After all these adjustments, the spring is as rigid as it was originally. These loop springs are not indicated in all cases of expansion, especially where a short spring is indicated. In these cases, a short finger is used.

Hooks for Intermaxillary Elastics.—Ofttimes, when intermaxillary elastics are used which are not under a great tension, they become unhooked when the jaws are at rest. The hook to prevent this is made as shown in Fig. 2. It is made from a 21-gauge spring wire which has been annealed. The hook is made in the shape of a question mark, with the end opposite that which is

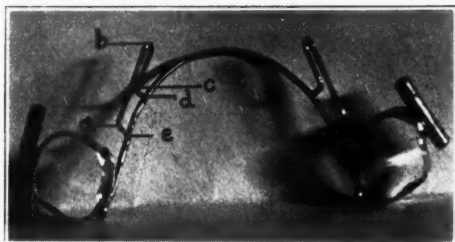


Fig. 1.—A lingual removable arch with lateral loop springs from a practical case.

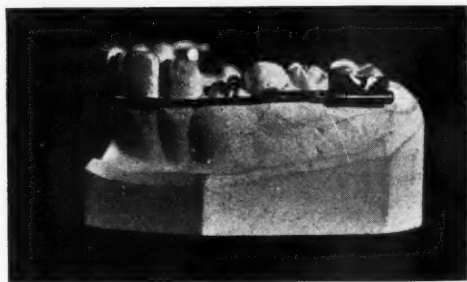


Fig. 2.—Shows the hook with the "balled" end used on the labial arch in connection with the intermaxillary elastics to prevent them from slipping off.

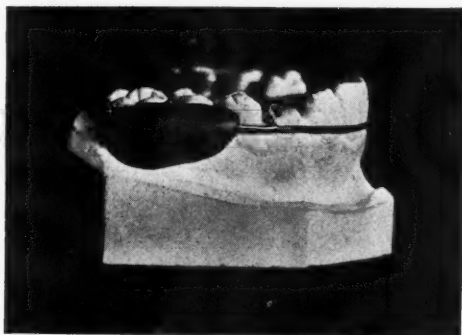


Fig. 3.—Shows how paraffin is applied to the labial arch and tubes to prevent irritation to soft tissues. Modeling compound is used here for photographic purposes.

to be soldered, and heated until the wire melts, forming a very small ball. It is left to cool, and the ball end bent very closely to the opposite side of the hook so that the rubber must be stretched to be hooked.

Use of Paraffin.—When appliances are set for the first time, very often there is much irritation from the buccal tubes. To overcome or prevent this irritation, the patient is instructed to soften paraffin or parawax in warm water and pack around and over the tubes, entirely covering them. This leaves a smooth surface for approximating the soft tissues of the cheek. Paraffin is sterile and consequently has no odors, and the tissues take kindly to it. Gum and cotton are neither sterile nor sanitary.

Use of Ligatures in Apical Movement of Anterior Teeth.—The direction of the apices of teeth bears a direct relation of the ligature to the teeth and

the wire to which they are ligated. It has been very noticeable in numerous cases that the roots of the four anterior mandibular teeth bear a related fan-shape appearance, the roots apparently converging toward the median line. As students were taught, the ligature should encircle the tooth to the distal, gingival to the arch wire and incisally to the arch wire on the mesial because of the tooth anatomy favoring ligature retention in that manner. This method of ligating results in an apical movement toward the median line. To correct this median position of the roots of the anterior teeth, one end of the ligature, after passing around the tooth, should cross the arch wire gingivally on the mesial, and incisally on the distal. (Only the lateral incisors are used to illus-

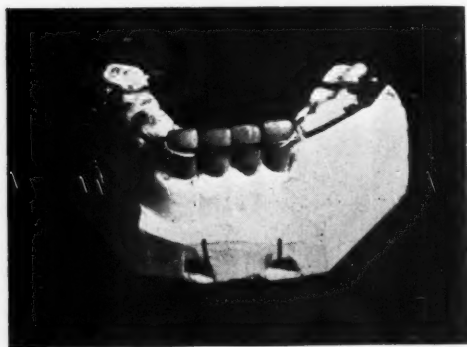


Fig. 4.—The ends of the wires directly below the lateral incisors indicate their apices, which are inclined toward the median line. The teeth are held in this position by a clamp located in back of the model, while they are ligated. After the ligatures are tied, the clamp is released to allow the force of the ligatures to move the teeth.

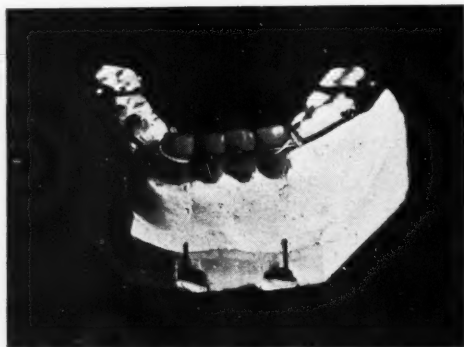


Fig. 5.—The spacing between the wires at the base of the models, Figs. 4 and 5, indicates the amount of apical movement, laterally, by ligating after this manner in Fig. 4.

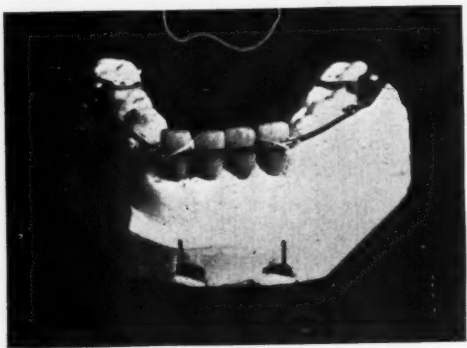


Fig. 6.—Ligatures placed the reverse as those in Fig. 4. The roots are in a comparatively vertical position.

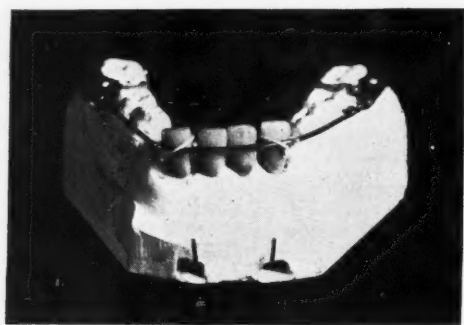


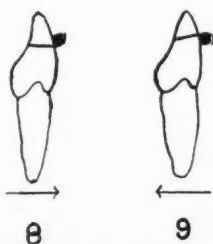
Fig. 7.—The "apical wires" indicate the root movement after ligating as in Fig. 6.

trate these points, Fig. 4.) Fig. 5 shows the result of ligating, in this manner, the ends of the wires directly below the crowns indicating the apices of the incisors. Using these same teeth, but reversing the position of the ligatures, the distal end crossing the labial arch (arch wire) gingivally, and the mesial end crossing incisally, the result in root movement will be a mesial movement of the apices and a slight distal movement of the crown. The crown will not move distally providing the four anterior teeth are ligated together with one ligature, interlacing same. A simple rule to follow is: if the distal end of the

ligature crosses the arch wire incisally, with the mesial end crossing gingivally, the root movement will be toward the distal, and vice versa. In short, distal-incisal, root moves distal; mesial-incisal, root moves mesial. If both ends of a ligature, after encircling a tooth, cross the arch wire (labial) on the gingival aspect, the root will take an anterior position, and if both ends of the ligature pass the labial arch on the incisal edge, the root will be moved lingually. (Figs. 8, 9.)

Heating the exposed ends of wires to a cherry red and allowing the metal to melt, forming a very small ball, makes a smooth end for approximating tissues.

All solder is used in wire form. The high karat in 27-gauge, and the easy



Figs. 8 and 9.—Arrows indicate the direction of roots when ligatures are placed as in the above figures, both ends of ligature passing gingivally and occlusally to the arch wire.

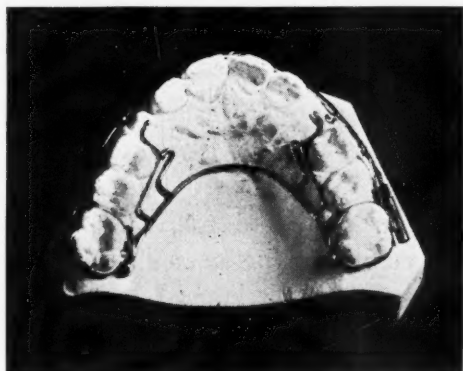


Fig. 10.—A combined labial and lingual arch with loop springs and finger springs on the anterior portion of the loop for purpose of guiding canines to position.



Fig. 11.—Shows a practical case in which loop springs were used for expansion. It also indicates how the loops are opened after first inserted as in Fig. 10, although not models of the same patient.

flow or low karat solders in 21-gauge. The solder comes in coils, which are dipped into S.S.W. soldering flux and heated to burn the vaseline off, leaving only the flux powder which adheres to the solder. This saves time, in that the solder and the part to be soldered need not be fluxed at each operation, and the solder, no matter how much is used at one time, is always clean after melting.

Both labial and lingual arches are adapted by heat. One end of the labial arch is inserted into one of the buccal tubes, and blowpipe heat applied where the wire is to be bent, adapting the wire where desired, finally approaching the opposite tube; here it is cut off, the end filed smooth, and inserted into the tube. Heat is again applied evenly over the entire wire

while both ends are in the tubes, thus reducing any spring which may not be desired, and at the same time tempering the wire by allowing it to air cool. The lingual arch is adapted in a similar way, using a slate pencil for detailed adaptation while heat is applied.

While soldering small wires such as finger and other auxiliary springs, it is desirable to have one, and probably two pairs of tweezers or soldering pliers, one pair, the ends of which are grooved, the groove being parallel with the handles, and the other, the groove being at right angles to the handles, and very close to the small end. The object is to prevent wires from twisting while holding with the tweezers. The grooves are made with a very fine knife-edge stone, making a shallow groove in each half of the small end of the tweezer, and coinciding. A twist drill of 24- or 25-gauge is firmly held in the grooves by squeezing the tweezers, and rotated either by engine or hand. This aligns the grooves. If the small end of the tweezer is large enough, it is convenient to have both grooves on one tweezer, which would save handling the extra pair.

When tying the first knots using silk or grass line ligatures, they can be held firmly while tying the second knot by having the patient wet the ligature with the tongue, and drawing the knot tight over the arch wire or other "high spot" to the desired tension. The ordinary knot will hold exceptionally well, and it is not necessary to twist the ligatures more than once. The second knot is made in the ordinary manner, and while the first and second knots are being drawn tightly together, they can be slipped either to the gingival or incisal of the wire, thus avoiding irritation to the cheek or lip. If the ligature is not wet, the knots will not slip, and must remain in the position where they were originally tied. Both ends of the ligature must be held taut, pulling slightly more on one, in the direction the knot is to be placed.

For convenience in having the right amount of air and gas in the orthodontic burner and blowpipe, two valves are connected between the air and gas supply pipes and the burner and blowpipe. One valve is used as a "shut-off" and the other as a "check." The "check" is set to allow the correct amount of air or gas needed for the burner or blowpipe, and need not be disturbed. The "shut-off" valve is the nearest the supply pipes and is used in its intended capacity, to stop the flow of gas or air. This arrangement of valves is convenient and saves time in that the air and gas need not be adjusted each time the burners are used. Most orthodontic burners have air and gas adjustments on them, but the above method prevents high air pressure on the hose, which is not desirable.

American Society of Orthodontists

The Twenty-fourth Annual Meeting of the American Society of Orthodontists will be held in the new Atlanta-Biltmore Hotel, at Atlanta, Ga., April 14, 15, 16 and 17, 1925. (Mark off the date now.)

Walter H. Ellis, Sec'y-Treas.,
397 Delaware Avenue,
Buffalo, N. Y.

Clinton C. Howard, President,
Doctors Building,
Atlanta, Ga.

OUTLINE OF THE FIRST INTERNATIONAL ORTHODONTIC CONGRESS

IN 1926, the American Society of Orthodontists will hold its twenty-fifth annual meeting, thus celebrating what we may consider the silver anniversary of the specialty of orthodontia. Not that the science of orthodontia was not both understood and practiced prior to that time, but it was approximately twenty-five years ago when orthodontia was launched as a scientific specialty.

Last March, as president-elect of the American Society of Orthodontists, I laid before that body the outlines of a plan for an International Orthodontic Congress in 1926, which had been a dream of mine for a number of years and which I had previously mentioned in a casual only to a very few friends. That plan (which I will outline also to you today) was to my pleasant surprise, cordially received by all present and endorsed most enthusiastically by many of the older and prominent men in the specialty. After much discussion it was voted by the American Society that the First International Orthodontic Congress be held in 1926, at New York City, and that the president, president-elect and secretary-treasurer of each orthodontic society in America (whose officers were then present) be constituted members of a committee on organization, and that the final organization of the congress be consummated at the next annual meeting of the American Society, to be held in Atlanta, Ga., April 14 to 17, 1925. It so happened that there was in attendance at this last March meeting of the American Society of Orthodontists at Kansas City, a majority of the officers of six orthodontic societies, namely: Pacific Coast, Southern, Southwestern, Rocky Mountain and New York Societies, and these officers all pledged their individual support and that of their society.

A chairman and a secretary were elected to serve until the organization meeting in Atlanta in 1925. As chairman, I was authorized to visit Europe this summer and if possible personally invite all the orthodontic societies of the several countries and the European Orthodontological Society to become a component part of the congress under the same conditions and terms as apply to the six societies in America.

ORGANIZATION

At the permanent organization meeting in Atlanta next April, a president-general, secretary-general and treasurer-general shall be elected by all those in attendance at the organization meeting, providing the orthodontic society of which they are a member has voted to become a component part of the congress.

The Board of Governors.—The board of governors shall consist of a representative from each component society together with the president-general, secretary-general and treasurer-general. This representative from each component society must be elected by the respective component societies not later

than June 1, 1925. In addition to their representative on the board of governors, each component society may elect from among its members one honorary vice-president of the congress and shall recommend to the board of governors not more than three names from which may be selected those men who in the judgment of the board have so contributed to the advancement and progress of the specialty as to warrant election to the position of honorary-president of the congress.

Duties of the Board of Governors.—The board of governors shall, as its name implies, be the governing body of the congress and in all matters pertaining to the advancement and conduct of the congress their decision shall be final. For the transaction of all business, seven shall constitute a quorum, but because of the probability that the members of the Board of Governors will be scattered over great distances in both America and Europe, an absent member may nominate a proxy for a particular meeting providing there is no objection from two or more members of the board. (This provision is merely inserted to expedite the transaction of business by the board of governors and to give its decisions a legal as well as an ethical standing.) The president-general may appoint with the consent of the board of governors such committees as will be found necessary to the successful consummation of the congress. The president-general, secretary-general and treasurer-general shall be members of the board of governors and members ex officio of all committees.

Committees.—Among the necessary committees will be an executive committee of five members who shall function for the board of governors and under its instruction. This executive committee will serve as an ad interim board and shall have all of the power and privileges of the board of governors, but its actions will be subject to the final approval or rejection by the board of governors itself. This committee will be found necessary to care for much detail work which it will be impossible for the board of governors to efficiently transact because of the aforementioned difficulty of obtaining frequent meetings.

There shall also be a program committee of five appointed by the president, three of whom shall be members of the Board of Censors of the American Society of Orthodontists. This committee shall have full charge of papers and clinics. The reasons for arbitrarily placing on the program committee three members of the Board of Censors of the American Society of Orthodontists are: that this committee always functions as a program and clinic committee for the American Society of Orthodontists; it is a progressing committee serving from year to year throughout a three year term and necessarily is familiar with that character of work. Further, inasmuch as the American Society is practically turning over to the congress its annual meeting of 1926, by including on the program committee the three members of the American Society of Orthodontists' Board of Censors, we will be able to avail ourselves of the material which has been collected during the preceding two or three years.

General business sessions shall be held during the first and last days of the congress, and at such other intervals during the congress as the officers

deem necessary. The greater part of the work of the organization of the congress, and of the congress itself must be done by the American members and a still greater part of the work must necessarily be done by those practicing in and near New York City acting on the several committees.

MEMBERSHIP

Membership in the congress shall be in two classes:

1. *Regular Membership*.—This membership can be had only through membership in component societies. It carries the right to vote and hold office. A payment of ten dollars (\$10.00) by a recognized orthodontic society and the endorsement of the plan of the congress makes that society a component part of the congress and its members regular members of the congress and entitles the society to a bound copy of the proceedings.

A regular member desiring a bound copy of the proceedings must pay ten dollars (\$10.00) as does a subscribing member.

2. *Subscribing Membership*.—This membership is open to all ethical members of the dental and medical professions, irrespective of their membership in the component societies. The dues for this membership shall be ten dollars (\$10.00) and shall entitle the member to all the privileges of the scientific session and a bound copy of the proceedings. He shall have no right to vote or hold office unless he is a regular member.

FUNDS

All monies, both from the component societies and the subscribing members, shall be received by the treasurer-general. No bills shall be paid by him until they have been O.K.'d by both the president-general and the secretary-general. The board of governors shall appoint an auditing committee of three members who shall audit the accounts of the treasurer-general after the close of the congress. Any monies remaining shall either be prorated by the board of governors to the various component societies in proportion to their membership in the congress or shall be held in trust by the treasurer-general or a designated bank or trust company until the second International Orthodontic Congress shall be instituted. Any deficits shall be met by the component societies in America in proportion to their respective membership in the congress.

It is probable that a word of explanation should be made as to why New York was selected as the meeting place of the congress. First of all from a geographic standpoint the eastern seaboard of the United States was considered the most convenient for both the American and foreign members. Inasmuch as New York is practically the port of entry to America of all steamship lines, those using steamship lines will look with favor on New York. Those using rail transportation in America will find New York most easy of access. Further, the question of accommodation is one that must be duly considered and there is no doubt but that New York can give equal, if not superior accommodations to that of any other city, and about as reasonable in price. From the standpoint of entertainment, New York will rank with London and Paris in the cosmopolitan variety of the entertainment which can be furnished

for the visiting members. From an educational standpoint New York is as favorable as any other city, and it doubtless has more orthodontists practicing in its confines than any other two cities in the world. It has, practicing within a radius of six hundred miles, probably as many orthodontists as in the rest of America. For this reason it will probably be possible to obtain a great wealth of clinical material and it will be further possible for the members of the congress to have the privilege of visiting the offices of more orthodontists than they could have if the meeting was held in any other city.

The latter part of June has been suggested as that time when the orthodontists can most conveniently leave their practices. Nearly all the schools of America close the latter part of May or the early part of June and the children will have left for their summer vacations. This time was also considered as a favorable date for the foreign members (and we expect a very free discussion of this particular point) because they could take advantage of the low rates on the westward trip and if they so chose could delay their return to Europe until the latter part of July when the low rates eastward would go into effect. It may be possible to obtain further lower steamer rates through the United States Shipping Board for those attending this congress.

William C. Fisher, Chairman.

St. Louis Study Club of Dentistry

At two o'clock on Saturday, March 21, 1925, at the Coronado Hotel, Spring and Lindell, the St. Louis Study Club of Dentistry will hold its seventh annual clinic, to which every ethical dentist is invited.

This institution, organized in January, 1919, is maintained for the purpose of teaching practicing dentists the latest and most modern developments in the science of dentistry. At the conclusion of each term, a clinic is held in order to show to the profession the results achieved in each of the subjects taught.

Inasmuch as neither the officers nor instructors receive compensation for their services, it is possible to conduct these classes without cost to the students.

The one hundred twenty-six students who were enrolled in the 1924-1925 term will participate in group clinics in the following subjects: Drawing; Dental Roentgenology; Fixed Bridgework; Porcelain Technic; Cavity Preparation and Casting Technic; Full Dentures; Root Canal Technic; Oral Diagnosis and Diseases of the Mouth; Conduction and Local Anesthesia; Operative Dentistry.

Following the clinic, a dinner, at which the members of the faculty will be the guests of honor, will be given. Ethical dentists are cordially invited to attend this function.

Bulletins, descriptive of the Study Club, may be had by addressing Dr. F. C. Rodgers, 309 Wall Building, St. Louis.

DEPARTMENT OF DENTAL AND ORAL RADIOGRAPHY

Edited By
Clarence O. Simpson, M.D., D.D.S., F.A.C.D.,
and Howard R. Raper, D.D.S., F.A.C.D.

A NEW KIND OF X-RAY EXAMINATION FOR PREVENTIVE DENTISTRY*

BY HOWARD R. RAPER, D.D.S., F.A.C.D., ALBUQUERQUE, N. MEX.

(Continued from January issue.)

VI

MAIN PURPOSE AND FREQUENCY OF RADIOGRAPHIC INTERPROXIMAL EXAMINATIONS

Briefly, but accurately, stated, the purpose of the radiographic interproximal examination is to prevent toothache, and so prevent the diseases which follow in the wake of toothache.

To accomplish this purpose the examination should be made periodically, so that all cavities will be found before they are large enough to involve the pulp. Just how often these periodic examinations should be made is a matter of which we will learn more as time goes on and as we use this interproximal examination more. As a profession, we have much to learn about the rapidity of the progress of decay.

I estimate that the examination should be made, say, yearly or bi-yearly, depending on the case. In an effort to be more specific let me say, once a year until the age of thirty; thereafter every eighteen months or bi-yearly, unless there is some special reason against this extension of the time of the interval. This suggestion is based on the fact that the average person is more immune to dental caries after about thirty years of age. There are other governing factors; no inflexible rule can be set down. The operator must use judgment.

How delightful it would be to practice this preventive dentistry. The names of patients should be kept in a suitable file so they may be notified when the time comes for their periodic interproximal examination. More than 95 per cent of toothache could be avoided in this way, and, so, more

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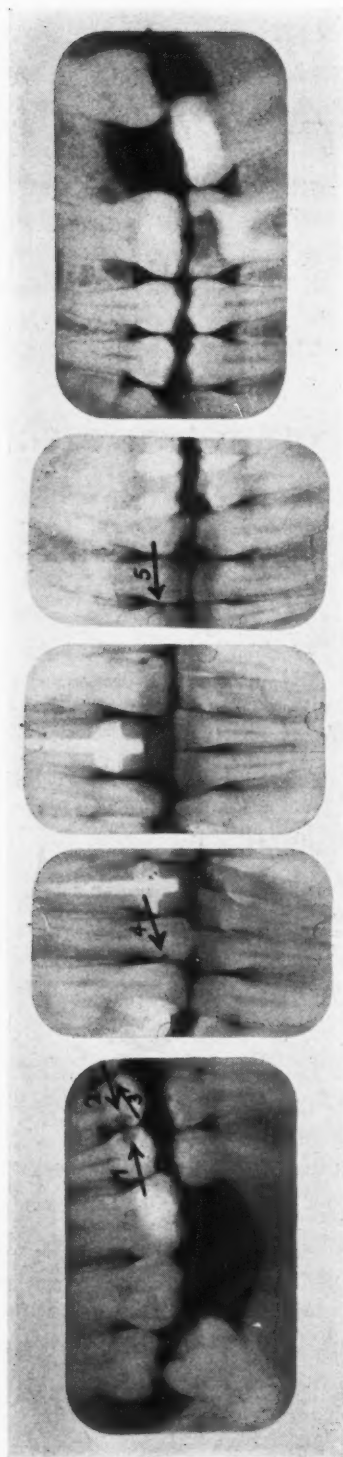


Fig. 6.—Arrows 1 to 5 inclusive all point to carious cavities. Some are very small (Arrows 3, 4 and 5) while in others the enamel has been completely penetrated and considerable underlying dentine destroyed.
 Note: Though not difficult to see in original negatives, small cavities are often indistinct due to loss of detail in halftone reproductions.

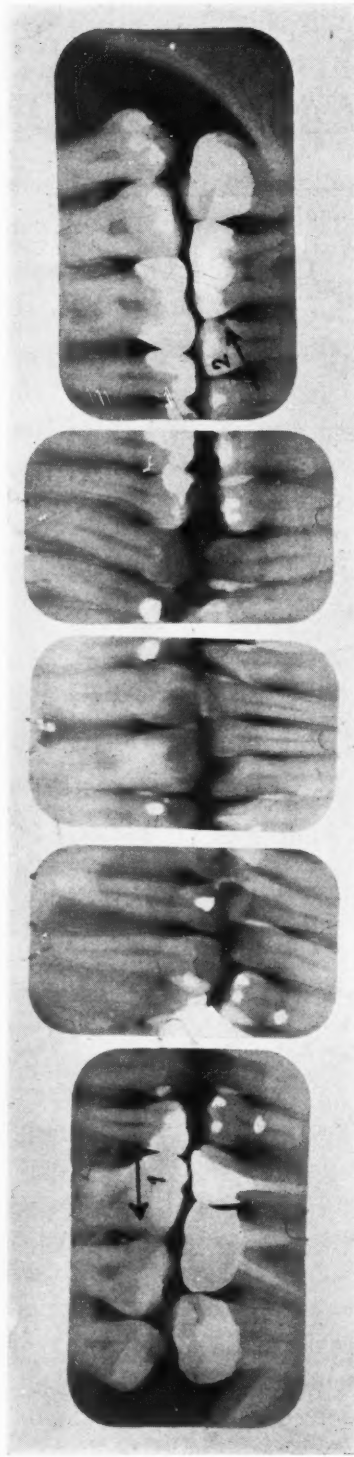


Fig. 7.—Arrow 1 points to a small cavity in the distal of the maxillary first molar. The enamel is just nicked on the distal of the mandibular second premolar, Arrow 2.

than 95 per cent of the kind of teeth (pulpless teeth) that cause systemic disease could be prevented.

Dr. C. O. Simpson has directed my attention to the fact that proximal decalcification can be detected before an actual cavity exists. Nevertheless the very small spot on the mesial of the central in Fig. 8 is an actual cavity.

When very small cavities are detected in the posterior teeth, it may not be advisable to fill them at once. It may be better to keep them under periodic observation at intervals of, say, six months, until they are of such size as to make filling advisable. (This will be discussed in detail later.)

VII

ADVANTAGES DERIVED FROM PERIODIC RADIOGRAPHIC INTERPROXIMAL EXAMINATIONS

The desirability of practicing preventive dentistry is something of which the dental profession is fully aware. This periodic interproximal examination is something tangible for both the profession and the laity to tie to. It is, I have said, "the peg" on which the new dentistry may be hung. Or, if you do not like that metaphor, then say it is the foundation on which the new preventive dentistry may rest.

Periodic interproximal examinations will keep the amount of tooth decay in the mouth at the lowest possible minimum at all times and in this way the aggregate amount of decay occurring is reduced. To make the meaning of that statement clearer and to give my own opinion added weight, let me quote one who is undoubtedly one of the most reliable writing men in dentistry, C. N. Johnson. I quote from a paper which appeared in the Dental Cosmos.*

"To detect cavities and fill them when they are small does something more than limit the decay in the teeth thus treated—it lessens the tendency to decay in other teeth in the same mouth."

VIII

THE FINANCIAL ASPECT

A further advantage of the interproximal examination is that it seems to be economically sound, from all angles. I can scarcely express my gratification at being able to say this. I have so often felt constrained to advocate things which were too expensive for the great mass of the common people. Aseptic canal surgery for example. But at last I am privileged to recommend something which is simultaneously a good thing for patients and also inexpensive. What a rare and pleasing combination.

People of the most ordinary means can afford to have the periodic interproximal examinations. In fact in the long run, the new preventive dentistry, based on the periodic interproximal examinations, will cost *less* than the old and still prevailing ridiculous practice of allowing teeth to ache, then trying to "fix" them.

*"Some of the Present Problems in Operative Dentistry," by C. N. Johnson. Dental Cosmos, October, 1921.

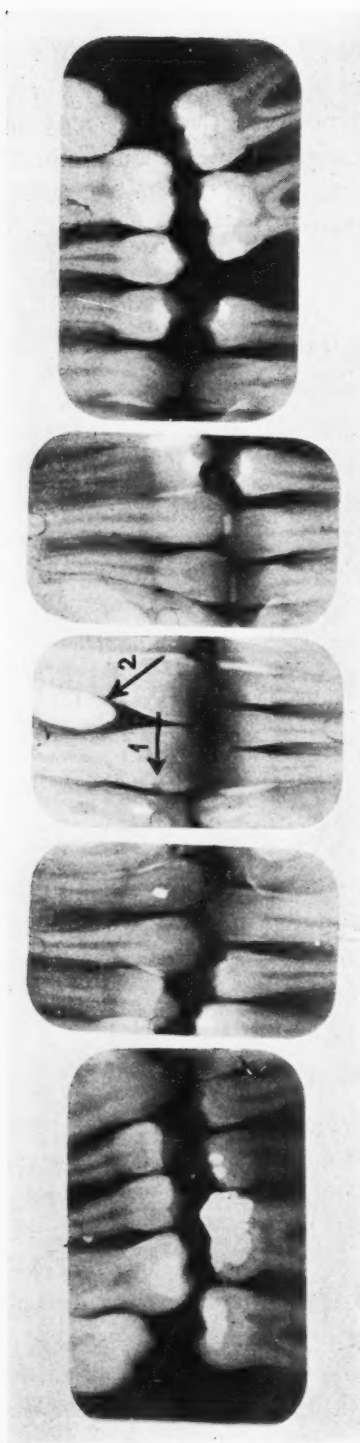


Fig. 8.—Arrow 1 points to a small cavity in the distal of the maxillary central incisor. Though small, an explorer could be passed into this cavity; it was through the enamel and attacking the dentine. It was filled. Arrow 2 points to a supernumerary tooth. It was something of a surprise to locate this tooth, by means of the interproximal examination; this case was one of the first few cases examined.



Fig. 9.—Arrow 1: Recurrence of decay at the cervical of the filling on the distal of the mandibular second premolar. There are many proximo-occlusal restorations in this mouth. Satisfactory examination of these many cervical margins is impossible save by the use of radiograms.

Arrows 2 and 4: Deep isolated pyorrhea pockets. (In the course of treatment a long flap of gum, which covered the pocket, indicated by Arrow 4, was removed, with gratifying results. In this case the gum was not dissected away; its removal was accomplished by cauterization with trichloroacetic acid.)

Arrow 3 points to a cavity in the mesial of the maxillary canine.

Let us consider very briefly the history of one tooth neglected until it aches. It is treated, the canals filled and the tooth crowned. Later the patient develops rheumatism. There are doctors' bills, druggists' bills, time lost from work. The tooth is radiographed. It is extracted, the bone curetted. A three-tooth bridge is made.

Think of how many periodic interproximal examinations and fillings could have been paid for with the money spent for pulp canal treatment, canal filling, crowning, extraction, curettement, the bridge, the physician's bill, the druggist's bill, and of how much more value such service is to the patient.

I have, in the past, often advocated a publicity campaign to spread the slogan, "Never Let Your Teeth Ache." But nothing much ever came of my efforts, one reason being the economic difficulties inherent to my proposals. The periodic interproximal examination will accomplish the same desirable prevention and it rests on a firm economic basis. That is to say, it will be paid for by the person benefited and the pay goes to the men who do the work. The enterprise does not depend for its success on sporadic philanthropy or individual sacrifice. It will develop naturally in the course of, and in harmony with, existing activities.

I am enthusiastic over the fact that poor people can afford the new preventive dentistry because, that being the case, they may get it. It has been my observation, loud and angry protests to the contrary on the part of the optimists notwithstanding, that in the end the people get only what they are able to pay for. Of course if they are not able to pay for the real thing, somebody will supply them with a cheap imitation, much to the damage of their health usually. But in the case of the coming new preventive dentistry, poor people can afford the real thing. (They cannot afford the real thing in the instance of aseptic canal surgery and practically never get it.)

The most important feature of the interproximal examination is, obviously enough, that it promotes the practice of preventive dentistry which will, unless all our calculations and theories are wrong, make human beings healthier; it will, to quote Dr. Mayo, "add ten years to human life."

I may say in passing, that the thought of adding ten years to life has little appeal for me. Life is long enough to suit me. But the thought of making life more livable while it lasts has a tremendous appeal. It would be a wonderful thing to accomplish. It is dentistry's great opportunity.

IX

SOME CLINICAL CASES

My research work in this field had a rather impressive (to me) beginning. The first negative made was of myself. It showed an *unsuspected* cavity. The next negative was of an associate, and it showed an *unsuspected* cavity! The next negative was of the office girl, and it showed an *unsuspected* cavity!! And the fourth negative made was of an office girl down the hall and it too showed an *unsuspected* cavity!!!

The girl from down the hall was referred to her dentist who at first

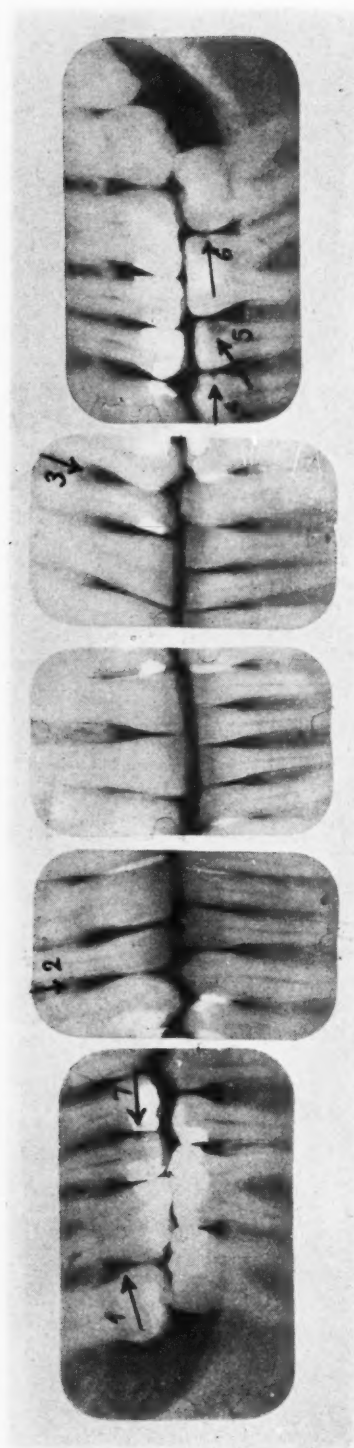


Fig. 10.—Arrow 1 points to a small bit of calculus on the mesial of the maxillary second molar. Arrows 2 and 3 point to incipient pyorrhea. Arrows 4, 5, 6 and 7 point to carious cavities. The cavity indicated by Arrow 5 is getting rather large, having destroyed considerable dentine; immediate filling is indicated. The cavities indicated by Arrows 4, 6 and 7 have just nicked the enamel; they should be kept under periodic radiographic observation.



Fig. 11-A.

Fig. 11-A.—Case in which the patient complained of pain "somewhere" on the right side. Proximal cavity suspected. Finding: No cavity but a metal filling entering pulp chamber of the maxillary second molar.



Fig. 11-B.

Figs. 11-B and 11-C.—Same case as Fig. 11-A but showing the periapical region of the maxillary second molar. Fig. 11-B fails to reveal the evidence of periapical osteoclasia (the malar shadow obscures the view) but Fig. 11-C, made at a different horizontal angle, reveals the lesion clearly enough.



Fig. 11-C.

declared there was no cavity. But, on more careful instrumental examination, the cavity was finally found, a very large one, in a lower molar, almost reaching the pulp.

Figs. 6 to 10 show full mouth interproximal examinations. Read the captions. The use of all five-film examinations here, to the exclusion of the seven-film examination, must not be taken to indicate that I use or recommend the five-film examination to the exclusion of the seven. What it does indicate is that, of the complete interproximal examination negatives at hand as I write, those of the five-film type seem better suited for halftone reproduction. The seven-film examination has not yet been given as much of a

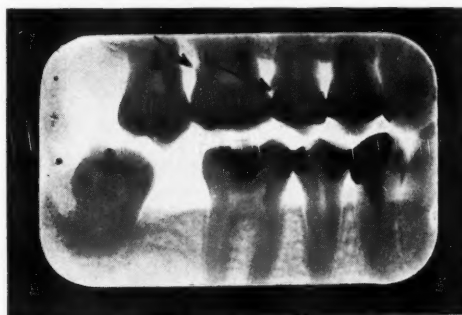


Fig. 12.—Case: Patient complained that sweet things hurt somewhere in the mandibular left posterior teeth. Findings: Cavity at cervical in maxillary second premolar (Arrow 1) and pulpless mandibular first premolar. The cavity was the cause of the symptoms. Arrow 2 points to a bit of calculus on the distal of the maxillary first molar.

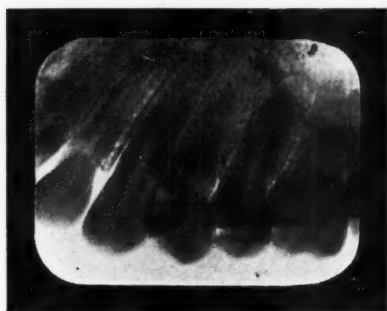


Fig. 13-A.



Fig. 13-B.

Fig. 13-A.—Shows what appears to be a cavity in the mesial of the first premolar. The spot, however, is not a cavity; it is the interproximal space superimposed on the mesial surface. The horizontal angle at which the radiogram was made was not correct; it was diagonally through the teeth, causing overlapping of proximal surfaces, instead of straight through as it should have been to get a good view of the interproximal spaces and proximal surfaces.

Fig. 13-B.—A case somewhat similar to Fig. 13-A. The radiolucent area on the mesial surface of the second molar was mistaken for a carious cavity. It is the interproximal space. The horizontal angle was not good, i. e., it was not straight through between the teeth. Note the overlapping of the distal of the second molar and mesial of the third molar. Such overlapping naturally obscures the view of both proximal surfaces. If the operator looks sharply, however, he may sometimes detect cavities, if they are present, in spite of this overlapping.

clinical tryout as the five-film examination. I suggest that it is perhaps best to start with the five-film examination and progress to the seven-film examination later if found desirable, just as the general radiodontic mouth examinations started with the ten-film survey and progressed to the use of fourteen or sixteen films.

As one would expect, a carious cavity or decalcified spot in the proximal surface of a tooth appears in the negative as a radiolucent (dark) area. One frequently sees the decay penetrating the enamel through a small hole, then spreading laterally, undermining the enamel, as is characteristic of the disease. When the operator detects the penetration through the enamel, he should scrutinize the underlying dentine carefully. Occasionally one may see the destruction of dentine going on under the enamel without being able to see where the enamel is penetrated. But care should be exercised not to mistake the normal radiolucency of dentine for a cavity (Fig. 14-A and 14-B); consider the shape of the radiolucent area and the appearance of other teeth in the same mouth.

While carious cavities in proximal surfaces are very easy to see in original negatives, it must be admitted—should be emphasized, in fact—that they may be difficult to see in halftones. This is due, of course, to the unavoidable loss of detail in halftones compared to original negatives.

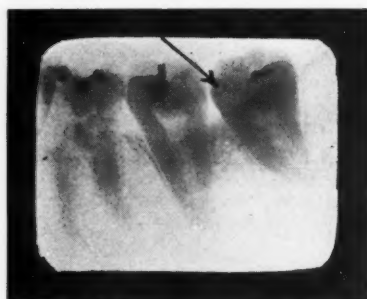


Fig. 14-A.

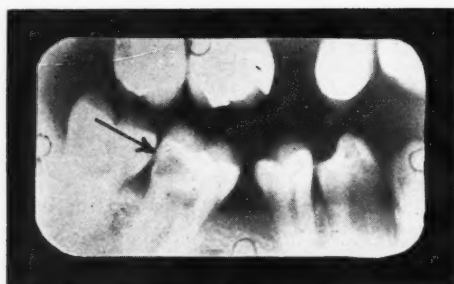


Fig. 14-B.

Fig. 14-A.—The triangular radiolucent area in the mesial of the second molar has somewhat the appearance of a cavity. It is not a cavity, however. The tooth has the appearance of having a cavity in it due to the contrast in shadow. The radiolucent area seems more radiolucent than it actually is because it is bounded by the comparatively radiopaque shadows of the external oblique ridge of the mandible and the enamel of the tooth.

Fig. 14-B.—The area, in the distal of the mandibular molar, indicated by the arrow, has a similar appearance to the area in the mesial of the second molar in Fig. 14-A. In this case, however the area does represent a large carious cavity. The radiogram does not show clearly where the decay penetrates the enamel; but it shows considerable loss of dentine beneath the unsupported wall of enamel. (The film used for the negative is too narrow and the teeth were not in good occlusion, so we fail to "get" the upper teeth. The negative was made long ago while the writer was still experimenting with holders and before the development of the bite-wing packet idea.)

Fig. 11-A was made for a lady who complained simply of "pain somewhere on the right side." Observe the filling material penetrating the pulp chamber. Pursuant to this finding Figs. 11-B and 11-C were made at slightly different angles, Fig. 11-C showing periapical osteoclasia.

Fig. 12 is of another case in which the lady complained that sweets hurt somewhere in the mandibular left back teeth. The radiogram shows a filling failing at the cervical in a maxillary premolar. This was the cause of the symptoms. Incidentally, Fig. 12 reveals the presence of an unsuspected pulpless tooth, the lower first premolar. A radiogram was made of this tooth in the usual manner showing the apex and apical tissues.

X

MISTAKES IN INTERPRETATION

Apparently there is a cavity in the mesial of the first premolar in Fig. 13-A. It is, however, the interproximal space superimposed on the mesial surface of the tooth. The horizontal (i.e., mesio-distal) angle at which the radiogram was made was wrong for getting a good interproximal view; that is to say, it was diagonally through the teeth instead of straight through between the teeth. (See also Fig. 13-B.)

Fig. 14-A shows an area of apparent radiolucency which might be mistaken for a cavity. This appearance is due to the heavy oblique ridges of the mandible. The tooth is not decayed. (See also Fig. 14-B.)

Silicate cement fillings cast so little shadow that it is impossible to differentiate radiographically between a cavity and a filling of this cement. If the cavity is somewhat geometric in outline this may be taken as a clue that the tooth is probably filled with silicate cement.

I have elsewhere (*Elementary and Dental Radiography*) enumerated a great number of mistakes in the interpretation of ordinary dental radiograms. There are comparatively very few mistakes one is likely to make in the interpretation of interproximal negatives. Their interpretation is indeed rather simple. That this is the case is indicated by an experience I have had repeatedly. After pointing out one cavity to a patient that patient has then said to me: "and there is another, and another," and so on, pointing out all the other cavities revealed in the examination.

(To be continued.)

DEPARTMENT OF ORAL SURGERY AND SURGICAL ORTHODONTIA

Under Editorial Supervision of

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A SYSTEMATIZED TECHNIC FOR THE REMOVAL OF IMPACTED MAXILLARY CANINES

BY LEO WINTER, D.D.S., NEW YORK, N. Y.

*Clinical Professor of Oral Surgery and Diseases of the Mouth,
New York College of Dentistry.*

SUCCESS in the removal of impacted maxillary canines without causing an irreparable injury, such as the loss of adjacent teeth, is dependent upon several important factors. Dental literature is replete with descriptions of various technics. However, in my experience, no one particular method is applicable to all cases. While no standardized technic can be established, owing to the varied positions which some of the teeth occupy, a more or less definite system of operation, based upon the following classification, will greatly facilitate the removal of these units.

Classification:

1. *Unilateral Impaction*—Teeth in arch.
Crown situated palatally, and lying close to the gingival margin.
2. *Unilateral Impaction*—Teeth in Arch.
Crown palatally and a distance from the gingival margin.
3. *Unilateral Impaction*—Edentulous Mouth.
No evidence by digital palpation of the position of the tooth.
4. *Unilateral Impaction*—Edentulous Mouth.
Evidence by digital palpation of the position of the crown palatally.

5. *Bilateral Impaction—Teeth in Arch.*

6. *Bilateral Impaction—Edentulous Mouth.*

When an impacted canine is discovered through routine radiography, and no septic disturbances are present, the patient is advised of the possibilities of orthodontic treatment; that is, provided the case presents favorable conditions; otherwise removal is advocated.

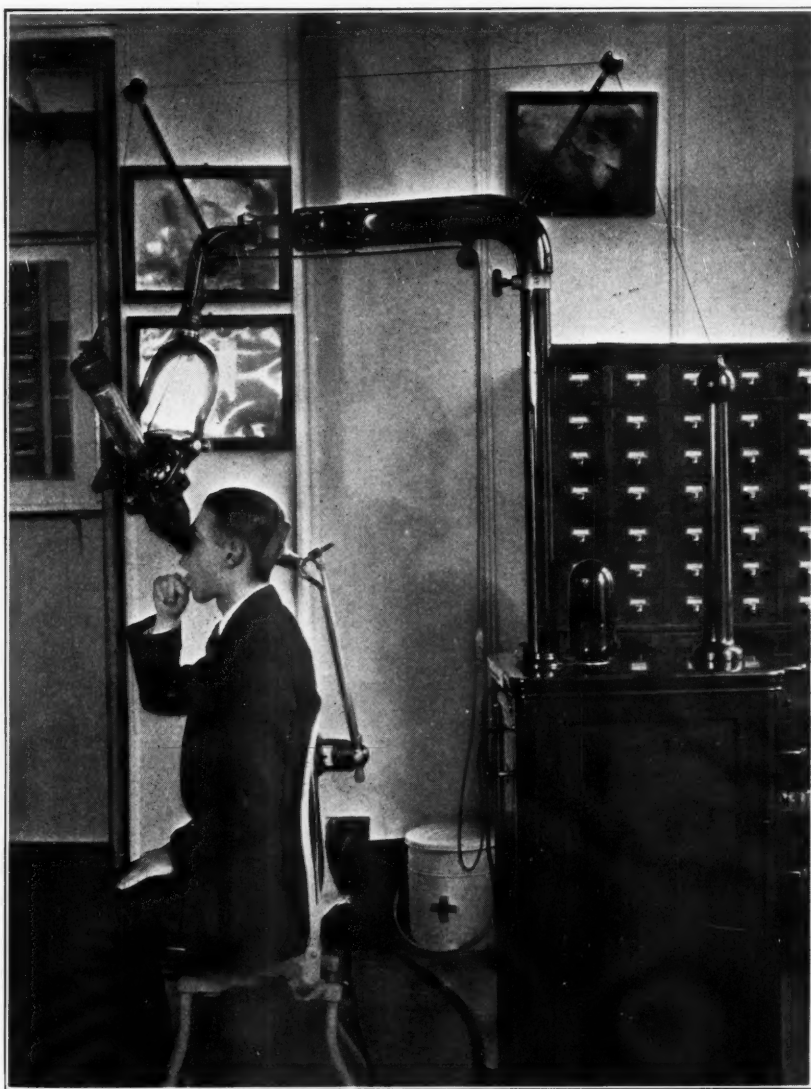


Fig. 1.—Position of tube in using No. 1 dental film. The resultant radiogram will show the relative position of the crown of the impacted tooth to the gingival margin.

The removal of the impacted canine is advised when the following conditions present themselves:

1. Prior to the insertion of a restoration in the maxillae, where all teeth are extracted with the exception of the impacted canines. (Pressure may cause injury.)

2. In cases of atypical neuralgia.

3. Patients who have systemic involvement of unknown origin.

In the latter instances, the patient should be informed that the tooth is removed only as a precautionary measure to eliminate all possible dental causes, because the radiogram may fail to reveal the presence of a cyst which usually envelopes the crown in this type of tooth.



Fig. 2.—Position of tube in using the No. 2 film. By this method an accurate localization of the impacted tooth is obtained.

RADIOGRAPHY

The first step in the removal of an impacted canine tooth is the careful study of radiograms. Two types should be taken: (1) The small dental film (No. 1); (2) The bite film (No. 2). Radiograms should show:

1. The location of the impacted tooth.
2. The position of this tooth.

3. The anatomic relations of the impacted tooth.
4. Type of tooth which we have to deal with, normal type or not.
5. The pathologic condition of the tooth and surrounding tissues.
6. The condition of the adjacent teeth.

The small dental film, when taken at the proper angle, shows the position the crown of the impacted tooth occupies with reference to the gingival margin (Fig. 1). This acts as a guide in determining the nature of the inci-



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.

Figs. 3, 4, 5 and 6.—Case in which roentgenologist made a written report of three impacted teeth. The large film is the view obtained by the overhead localization method, showing but one tooth impacted.

sion that is to be made. This form of film, however, is likely to be very misleading in the localization of the impacted canine. It might appear from this character of picture that the tooth is very superficially located; whereas in the majority of cases it is found to be very deep.

The ordinary x-ray film fails to show whether an unerupted or an impacted tooth is lying buccally or lingually. A stereoscopic radiogram would

show the exact location, but the time and great care required by this technic has made it unpopular.

The best means of radiographic localization is to direct the x-rays in such a manner that a cross section of the tissues under examination will be obtained. This is done by placing a film in the mouth and by having the patient bite down upon it. The film thus rests between the maxillary and the mandibular arches. If the patient has a marked overbite, which tends to bend the film, it is best to place a piece of metal under the film to keep it straight. The rays are directed approximately at right angles to the film and parallel with the long axis of the teeth. The point of entrance of the

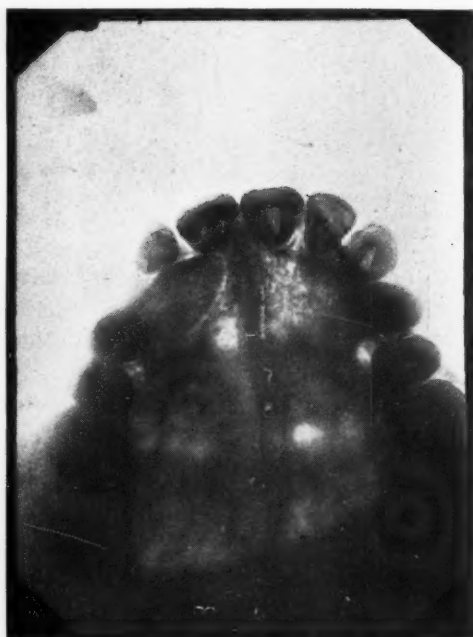


Fig. 7.



Fig. 8.

Fig. 7 and 8.—Comparison of radiograms of the same case. The small film (No. 8) is misleading for localization. The impacted tooth in this picture appears to be very superficial. The large film (No. 7) shows the true position of the impacted tooth to be diagonal, the crown portion toward the palate, the apex toward the buccal.

central x-ray is right through the frontal bone, at and between the frontal sinuses (Fig. 2).

Adjusting the tube in this position instead of at the top of the head, places this technic within the reach of the average dentist. When the rays are directed through the top of the skull the machine must be backing up $4\frac{1}{2}$ to 5 inches of spark-gap. The machines in the hands of most dentists are of the $3\frac{1}{2}$ inch, 10 milliamperes variety, sufficient for the first method described, but will not be sufficient when used for the position described by Dr. C. O. Simpson. It can readily be seen how this manner of localization works to the advantage of most of the dental profession, in using the $3\frac{1}{2}$ inch 10 M.A. machine at a target film distance of 15 inches with an extra fast film. We would expose from 8 to 10 seconds.

When it is found through radiographic findings that the pathologic con-

ditions present make it necessary to remove other teeth with the impacted canine, the patient should be so advised. It is best that the tooth or teeth be removed at the same sitting, rather than at a subsequent operation. Very often this procedure facilitates the operation. It must, however, be made clear that it is not necessary to remove any sound tooth or teeth for the purpose of gaining access to the impacted tooth.

The use of only a small film at different angles is apt to portray an erroneous outline of the crown portion, so much so that it may be interpreted as another tooth. I cite a recent case: A patient was referred to me for the removal of three impacted teeth (Figs. 3, 4, 5). The patient presented radiograms with a written interpretation. My conclusion, after examining the

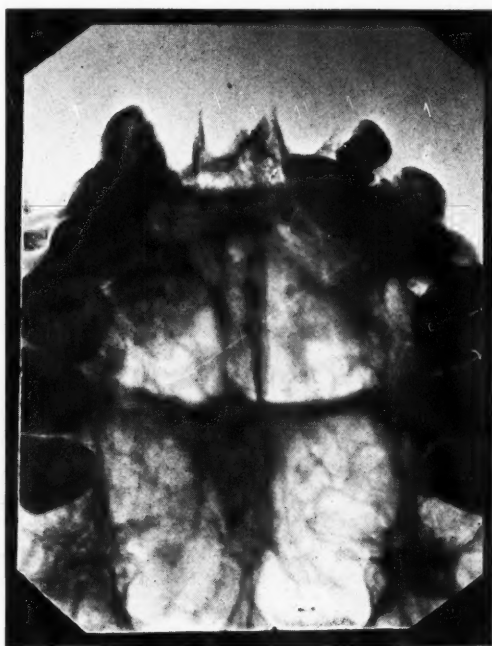


Fig. 9.



Fig. 10.

Figs. 9 and 10.—Comparison of the two forms of radiograms taken on a dry specimen.

radiograms, was that only one tooth was impacted. In order to confirm my judgment, a No. 2 bite film was used as previously described. The radiographic finding verified my decision (Fig. 6).

At a recent oral surgery clinic, the clinician, prior to the operation, cautioned the audience against attempts to operate without good radiograms. However, he used only a small film and, after twenty minutes of vain effort on the right maxillae, he decided to open the left side and there lay the impacted tooth.

In speaking of the impacted teeth lying buccally or palatally reference is made to the crown position only. In practically all cases the impacted tooth lies diagonally, the crown portion toward the palate, and the apex toward the buccal surface. (Figs. 7 to 19.)

TECHNIC OF OPERATION

Asepsis and anesthesia are vital factors in the success of any operation. Our aim is to secure the highest degree of asepsis that is humanly possible, although in oral surgery this cannot be maintained analogous to abdominal operations. Even though many operators think they can disregard asepsis because the oral cavity contains innumerable bacteria, we must, however, prevent the introduction of foreign bacteria.

Sterile gauze and sterile linen are as essential as sterile instruments. The use of absorbent cotton, shaped into sponge forms, rather than the use of sterile gauze, is contrary to correct surgical procedure.



Fig. 11.



Fig. 12.

Figs. 12 to 19.—Showing cases in which because of the pathology present, teeth in addition to the impacted canines had to be removed in order to clear up the diseased areas. A careful study of the radiograms will reveal just which tooth or teeth it will be necessary to remove. This should be done as a part of the operation for the removal of the impacted tooth.

PREPARATION OF THE FIELD OF OPERATION

The technic consists first in spraying out the mouth with an antiseptic solution; the lip is then held away from the gum, and with sterile gauze sponges wipe all mucus from the field of operation. Then, with a cotton applicator dipped in a solution of aconite and iodine, paint the area which you expect to inject into and upon which you purpose operating.

ANESTHESIA

Novocain-Suprarenin is unquestionably the best anesthetic which we can use in operations of this nature. Particular attention should be paid to the paraphernalia for producing anesthesia. A sterile solution, a sterile syringe,



Fig. 13.



Fig. 14.



Fig. 15.



Fig. 16.



Fig. 17.

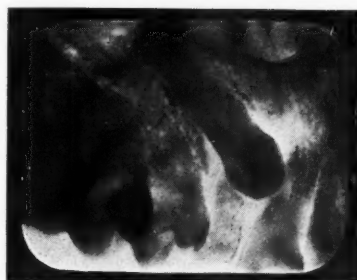


Fig. 18.

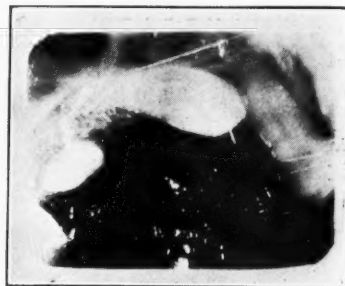


Fig. 19.

and a sterile needle will obviate any possibility of needless suffering to the patient and will relieve the operator of considerable anxiety.

By the use of a sterile solution of Novocain-Suprarenin the continuity of surgical asepsis above mentioned is assured. By its use, we obtain a comparatively ischemic area, more pronounced with the "E" than with the "T" formula. In using local Novocain anesthesia we obtain the cooperation of the patient which is a valuable adjunct.

A 2 per cent solution of Novocain-Suprarenin may be used with perfect



Fig. 20.



Fig. 21.

Figs. 20 and 21.—Overhead localization. Two radiograms showing the accurate position of the impacted tooth. It may be clearly seen from these pictures that the crown of the impacted tooth lies palatally, the root portion cannot be clearly discerned for it is situated more interiorly toward the buccal aspect.

safety, although it will be found that in the hands of the experienced operator lesser concentrations from 1 per cent to 1½ per cent will give equally perfect anesthesia.

An injection into the anterior palatine foramen, with slight infiltration in the region of the impacted canines and first premolars, augmented by infiltration of the buccal aspect on each side, will insure perfect anesthesia. If the patient is nervously inclined, the administration of camphorated validol, 7 to 8 gtt., fifteen to twenty minutes prior to the operation will generally quiet him.

(To be continued)

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EDITORIALS

The First International Congress

IN this issue of the INTERNATIONAL JOURNAL OF ORTHODONTIA, ORAL SURGERY AND RADIOGRAPHY will be found an "Outline of the First International Orthodontic Congress," as was officially presented to the New York Society of Orthodontists by Wm. C. Fisher, President-Elect of the American Society of Orthodontists and Chairman of the Organization Committee of the First International Orthodontic Congress.

This preliminary outline is the result of much thought and study by Fisher and has been drawn up to meet the conditions which exist in the orthodontic profession in America and Europe.

It is needless to say that we are in hearty accord with the idea of the

Orthodontic Congress, and we believe that the plan as published is the best that can be worked out to meet the needs of the various societies.

The only part of the outline over which there might be any misunderstanding is that which relates to the two classes of membership. It will be noticed that regular membership to the congress can be obtained only through component societies. Each society, by paying ten dollars to the congress, makes all of its members regular members of the congress with the right to vote and hold office; but a regular member will not obtain a bound copy of the proceedings of the congress unless he also pays ten dollars. Undoubtedly, many members of component societies will be anxious to receive a copy of the proceedings and will readily contribute the required amount. Some may raise the question as to why a plan was suggested whereby every member of the component societies becomes a regular member of the congress by the society paying the small sum of ten dollars. It is our belief that this plan of membership was suggested by the chairman of the organization committee after a careful consideration of conditions as they exist in Europe. It must be remembered, the question of foreign exchange is by no means satisfactorily settled. This may be illustrated by the man who earns francs and then spends dollars; his earnings are greatly diminished as soon as he starts to spend in another country. It would be practically impossible for the European orthodontic societies to become component parts of the congress except by allowing the societies to obtain membership upon the payment of ten dollars.

Another thing to remember in regard to individual members and those contributing ten dollars in order that they may receive the Proceedings of the Congress, is that a great many orthodontists in America are members of more than one orthodontic society. For example: nearly everyone who can qualify for membership in the American Society of Orthodontists, is a member of that society and he is also a member of the local orthodontic society, or of a sectional orthodontic society where he resides. The plan of the organization committee is to request the American Society of Orthodontists, at the meeting in April, to take such action as will be necessary to authorize its treasurer to remit to the organization committee of the Orthodontic Congress, ten dollars for each active member in the American Society of Orthodontists in good standing in 1925. If this plan is followed, it will be readily seen that it will produce an overlapping of membership, if each member of the component societies in the United States who is also a member of the American Society of Orthodontists is requested to contribute ten dollars, as each member of the American Society of Orthodontists will do. However, we believe that every member of a component society who is not a member of the American Society of Orthodontists will desire a copy of the Proceedings of the Orthodontic Congress. Therefore, the best plan to follow would be one which was adopted by the New York Society of Orthodontists, whereby its treasurer was instructed to pay ten dollars to the organization committee of the Orthodontic Congress for each member of the New York Society of Orthodontists who is not a member of the American Society of Orthodontists. This action was taken with the belief that the

American Society of Orthodontists, at their meeting in April, will follow the plan of membership which is outlined by the chairman of the organization committee.

The subscribing membership needs little discussion as it is very clearly outlined in the preliminary report published in this issue of the Journal. It is provided so that men who are not members of the American Society of Orthodontists or a component society can receive the bound copy of the proceedings if they so desire. Subscribing members cannot vote or hold office or take part in the business session.

Another portion of the outlined plan to which we wish to call attention, relates to the board of governors. Each component society will have a representative on the board of governors, who must be elected not later than June 1, 1925. A number of the component societies in America will not have a meeting prior to this date, after the meeting of the American Society of Orthodontists in April. It would, therefore, be wise for these societies to elect a member of the board of governors at their last meeting held before June 1. This action is suggested to enable the board of governors to be formed at an early date.

In the outlined plan as presented by the chairman of the organization committee, we learn that New York has been selected as the meeting place, and the latter part of June has been suggested for the date of the meeting. There has been considerable opposition in years past to the American Society meeting in New York because it necessitated such a long journey for many members; however, it must be remembered that the meeting of the Orthodontic Congress is international in scope and the foreign members must be given consideration. The third week in June, owing to climatic conditions, would be the most favorable time. However, it must be remembered that the date has not been definitely fixed, as conditions may arise which will make it desirable to select a later date.

It is hoped that the various members of the component societies will study the plans for the congress so that when they are considered by the society each member will be able to discuss the plans in detail. Any suggestions made in the way of changes should be advanced for the welfare of the congress as a whole rather than for the welfare of the individual. If every member of the component society will keep these points in mind, we think that the officers of the International Congress will be able to produce the greatest orthodontic meeting ever held.

Southwestern Society of Orthodontists

The next annual meeting of the Southwestern Society of Orthodontists will be held in Tulsa, Oklahoma, April 8 to 11, 1925. An excellent program has been arranged and a one hundred per cent attendance promised. Most of the members are planning to take a special car from this meeting to the American Society meeting in Atlanta, and the committee promises a very enjoyable trip enroute.—P. G. Spencer, Secretary, Amicable Bldg., Waco, Texas.

ORTHODONTIC NEWS AND NOTES

New York Society of Orthodontists

The Annual Meeting of the New York Society of Orthodontists will be held the afternoon and evening of Wednesday, March 11, 1925, at the Hotel Vanderbilt, Park Avenue and Thirty-fourth Street, New York City.

In addition to an excellent scientific program an important business session will be held at two o'clock.

Election of officers for the ensuing year and election of representatives for the First International Orthodontic Congress will take place. Also space in the American Society of Orthodontists' special train for Atlanta, Georgia, will be opened for reservations.

William C. Fisher, Secretary.
501 Fifth Avenue,
New York, N. Y.

The First District Dental Society, State of New York

The First District Dental Society, State of New York, extends a cordial invitation to all members of the American Dental Association, to attend its annual meeting on Monday, March 2, 1925.

The program is being arranged to cover every phase of modern dental practice, upon which demonstration clinics and lectures will be presented. It is desirable that members and guests, who expect to attend this meeting, register with the general secretary, Miss E. M. Davis, 250 West Fifty-seventh Street, in order that suitable reservation may be made for attendance at the clinics.

The clinics will be held at the Infirmaries of Columbia University Dental School, New York College of Dentistry and other hospitals and infirmaries. The paper of the evening will be given at the Academy of Medicine at 8 P.M.

Full detailed programs and cards of admission will be issued on application.

CLINICS FROM 10 A. M. TO 5 P. M.

Progressive Clinic by the Ceramic Clinic Club of New York City.

Indications for the porcelain jacket crown; studying the case. Milton Cohen.

Preparation for the shoulder and shoulderless jacket crowns. Carlisle C. Bastisn.

Impression taking; die making; model mounting; the making of the matrix. Oscar J. Chase, Jr.

The method of reproducing the natural teeth shades; packing, moulding, and carving of porcelain to individual case requirements. William J. Hoag.

The baking and staining of porcelain. Charles B. Mandelbaum.

Finishing the crown; cementation. William A. Squires.

PROSTHODONTIA

An improved technic of swedging metal bases which overcomes the inaccurate fit due to shrinkage of zinc and Babbitt metal dies. Russell W. Tench.

1. An improved buccolingual attachment.

2. Designing of saddles and partial dentures illustrated on models from practical cases. A. Gueft.

1. Demonstration in the reproduction of tooth forms.

2. Demonstration in the construction and completion of a simple Chayes' bridge.

M. Diamond.

Closed mouth impressions and anatomically occluded dentures. M. Schechter.

ORAL SURGERY AND EXODONTIA

Fractures. Theodore Kaletsky.

The importance of Roentgen Examinations in Oral Diagnosis. Sidney Riesner.

Root Amputations. Herman L. Reiss.

Malposed Teeth. M. L. Rosoff.

The Importance of Pathological Tests in Oral Diagnosis. Nathaniel Freeman.

Osteomyelitis. Theodor Blum.

Rational treatment of Pulpless Teeth. E. Alan Lieban.

Operation for the removal of Dentigerous and Follicular Cysts under Novocain Anesthesia. Leo Winter.

Removal of Impacted Mandibular Third Molar. David G. Baker.

Alveolectomy. N. Pollinger.

The Principles of Instrumentation in Surgery of the Root Surface. John Oppie McCall.

Unerrupted and impacted teeth and postoperative treatment. B. B. Palmer.

Cases as they present for operation. Leo Stern.

Block Anesthesia. Harry M. Moss.

Clinic Cases as Present. Adolph Berger, Harry Dunning, F. McCaffrey and Douglas Parker.

General Anesthesia (Nitrous Oxide and Oxygen). Harry M. Seldin.

Removal of Impacted Mandibular Third Molar. Charles Vetter.

Selection of an Anesthetic in Oral Surgery and Exodontia. Michael M. Moss.

ORTHODONTIA

Photographic Techniques, including Facial and Cranium Measurements. Joseph Stahl.

Plaster Impression Technique. Harry E. Abelson.

Clinical Diagnosis of Malocclusion. Martin Dewey.

Practical Orthodontia cases under Treatment. Martin Dewey, Josephine Abelson.

Some typical Malocclusions. Ralph Waldron and Julius Goldberg.

Radiographic Interpretation and Early Diagnosis of Malocclusion. James C. Allan.

DENTAL HISTOLOGY

Practical and Microscopical Demonstration of the Existence of an Organic Matrix in the Enamel Organ of the Human Teeth. Charles F. Bodecker.

Demonstrations. J. W. Dickinson, LeRoy Hartman and Assistants, Ellison Hillyer and Assistants.

PROSTHODONTIA

1. The use of stones (a new precision abrasive) in cavity preparation.

2. The demonstration of new precision tools and vibrationless hand pieces, which, in conjunction with the stones, will enable the dentist to prepare his cavities with the least

annoyance to himself and the patient. The possibility of keeping these hand pieces absolutely clean and sterile.

3. The demonstration of detailed steps in the construction of a Chayes' movable, removable bridge, the use of the new student set of instruments for the purpose of paralleling the attachments for such bridgework.

By A. Roney, J. Metger, M. L. Perlman and E. Pickhardt.

PATHODONTIA

Clinics will be given by the various members of the Pathodontia Section on the subjects of Root Canal Technique.

1. Cultures of Root Canals.
2. Root Canal Filling.
3. Callahan and Rhein Methods.

Restorations in Pyorrhea. J. K. Burgess.

Replantations. H. Tratner.

Plates and Bridge Work. Chas. Asch.

Steel Plates (German Method). Richard Blum

Immunization. Kaufer.

Root Canal. J. Schneer.

Cavity Preparation and Inlays. I. A. Press.

Pinlay attachments. Henry Wasserman.

ORAL HYGIENE

Demonstrations showing the work of the Oral Hygienist in the Public Schools and Clinics of New York City.

OFFICE ROUTINE

By the Educational and Efficiency Society for Dental Assistants.

SCIENTIFIC SESSION

The essayist of the evening will be Herman E. S. Chayes, whose paper is entitled, "The Practical Solution of Difficult Dental Problems."

This paper will be read at the Academy of Medicine, 17 West 43 Street at 8 P. M.

Synopsis: The Plan, the Starting Point, the Destination. The means to the end.

The mathematical factor.

The biologic factor, as expressed in the neural and nutritional needs of the case.

The physiological factor.

The anatomical factor.

The hygienic factor.

The integration of all the factors in their proper interplay to produce an acceptable compliment to the remaining dental organs in the case to be restored. Illustrated with lantern slides, radiograms and original drawings.

Formal Discussion by Charles Vetter, M. Diamond and R. Ottolengui.

Executive Committee of First District Society.

Lewis K. Mobley.

James C. Allan.

Armin Wald, Chairman.

40 EAST FORTY-FIRST STREET.

Tennessee State Dental Association

The Fifty-eighth Annual Meeting of the Tennessee State Dental Association will be held in Knoxville, Tennessee, May 5, 6, and 7, 1925.



Dr. Clinton C. Howard, Atlanta, Ga.

President of the American Society of Orthodontists, 1924-1925.